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# PEDS

5

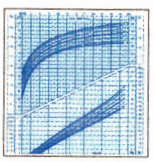
FIFTH EDITION 2012/2013

## PEDIATRICS BOARD REVIEW

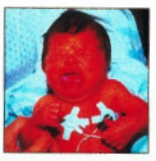
# CORE CURRICULUM

BOOK 1

GROWTH AND DEVELOPMENT / PREVENTIVE PEDIATRICS



COMMON PEDIATRIC DISORDERS



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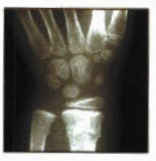
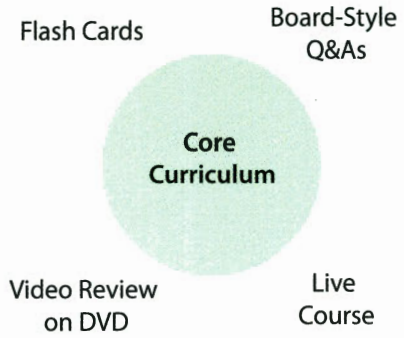


ADOLESCENT HEALTH AND GYNECOLOGY

# Welcome to a flexible, synergistic Board-prep learning system.

## Start with the Core Curriculum and build the study/review combination that works for you!




Our study/review system consists of diverse content geared to specific learning goals. The components work synergistically, to create a powerfully fused body of relevant, retained knowledge. A variety of content delivery formats also allows you the flexibility to build a system that fits your own study mode preferences. You ideally begin with the Core Curriculum, the foundation resource with comprehensive coverage of all relevant topics in Pediatrics. Then you can enhance this Core learning with one or more of the following: • Pediatrics flash cards to hone disease/syndrome differentiation skills • Pediatrics Board-Style Questions & Answers for realistic self-testing and knowledge assessment. • Video Board Review of Pediatrics on DVD for dynamic audio-visual presentation of Board-relevant topics. • A live Pediatrics Intensive Board Review Course where nationally recognized subspecialty experts teach you their specific topics. Using these resources, you can design strategies for study and review that capitalize on the individual and combined strengths of each component and that fit your learning style preferences.

## A look inside the Core Curriculum — and how it works for you

**Clean and clear organization:**
A logical sequencing of major and supporting topics guides you through the material and enhances your understanding of topical relationships.

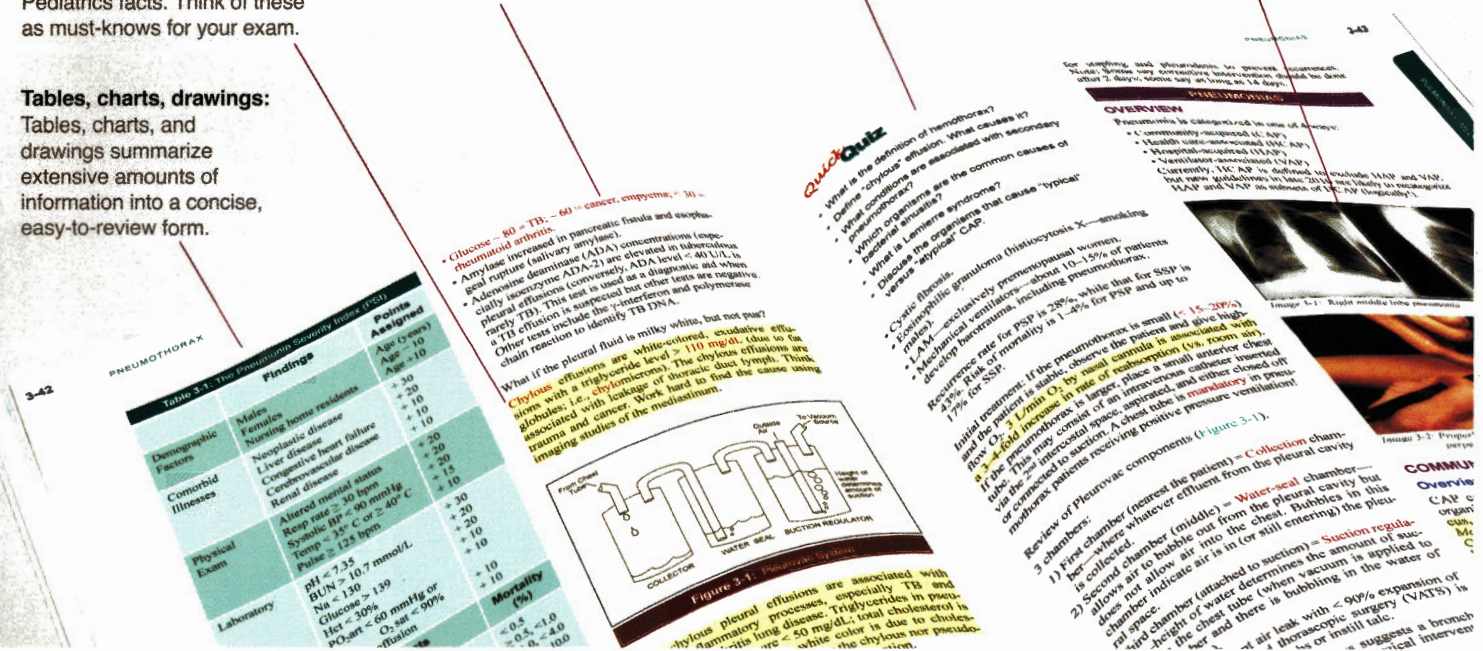
**Must-know highlights:**
We've highlighted for you in yellow the most fundamental Pediatrics facts. Think of these as must-knows for your exam.

**Tables, charts, drawings:**
Tables, charts, and drawings summarize extensive amounts of information into a concise, easy-to-review form.

**Accent type color:**
Burgundy type applied to selected terms and phrases gives a tonal emphasis to the text, much the way a teacher would use inflection in a great lecture. This also alerts you to key facts and fine points of distinction.

**Quick Quizzes:**
On every 2-page spread you'll find short questions to test yourself on what you've read. The answers to these questions will be found in the yellow-highlighted text, in the tables and charts, or in other graphics.

**Medical images:**
Photos, scans, x-rays, scopes and other images give visual clarification and emphasis to the text.



| ABP Topic | Core Book # | Look in these sections of the MedStudy Pediatrics Board Review Core Curriculum |
|---|---|---|
| Growth & Developmental Milestones | 1 | Growth & Development/Preventive Pediatrics; Common Pediatric Disorders; Adolescent Medicine & Gynecology |
| | 3 | The Fetus & Newborn |
| | 5 | Endocrinology |
| Nutrition & Nutritional Disorders | 4 | Gastroenterology & Nutrition |
| | 1 | Common Pediatric Disorders |
| | 5 | Hematology |
| Preventive Pediatrics | 1 | Growth & Development/Preventive Pediatrics; Adolescent Medicine & Gynecology |
| | 2 | Infectious Disease |
| Poisoning & Environmental Exposure to Hazardous Substances | 1 | Growth & Development/Preventive Pediatrics; Common Pediatric Disorders; Emergency Pediatric Care; Adolescent Medicine & Gynecology |
| Fetus & Newborn Infant | 3 | The Fetus & Newborn |
| | 1 | Growth & Development/Preventive Pediatrics; Common Pediatric Disorders |
| Fluid & Electrolyte Metabolism | 4 | Gastroenterology & Nutrition |
| | 5 | Nephrology |
| | 3 | Metabolic Disorders |
| Genetics & Dysmorphology | 3 | Genetics; The Fetus & Newborn |
| Allergic and Immunologic Disorders | 2 | Allergy & Immunology |
| | 4 | Respiratory Disorders |
| Infectious Diseases | 2 | Infectious Disease |
| | 3 | The Fetus & Newborn |
| | 1 | Adolescent Health & Gynecology |
| Metabolic Disorders | 3 | Metabolic Disorders |
| Endocrine Disorders | 5 | Endocrinology |
| | 3 | Genetics |
| | 1 | Adolescent Health & Gynecology |
| Gastrointestinal Disorders | 4 | Gastroenterology & Nutrition |
| | 1 | Common Pediatric Disorders |
| Respiratory Disorders | 4 | Respiratory Disorders |
| | 2 | Allergy & Immunology |
| Cardiovascular Disorders | 4 | Cardiology |
| | 3 | The Fetus & Newborn |
| Blood and Neoplastic Disorders | 5 | Hematology, Oncology |
| Renal Disorders | 5 | Nephrology |
| | 3 | The Fetus & Newborn |
| Genital System Disorders | 5 | Nephrology; Endocrinology |
| | 3 | The Fetus & Newborn |
| | 1 | Growth & Development/Preventive Pediatrics |
| Neurologic Disorders | 3 | Neurology; Genetics; Metabolic Disorders |
| | 2 | Infectious Disease |

| ABP Topic | Core Book # | Look in these sections of the MedStudy Pediatrics Board Review Core Curriculum |
|---|---|---|
| Musculoskeletal Disorders | 1 | Common Pediatric Disorders |
| | 5 | Rheumatology |
| Skin Disorders | 2 | Dermatology; Infectious Disease |
| Collagen Vascular & Other Multisystem Disorders | 5 | Rheumatology |
| Disorders of the Eye | 1 | Common Pediatric Disorders |
| Ears, Nose & Throat Disorders | 1 | Common Pediatric Disorders |
| | 2 | Infectious Disease |
| | 4 | Respiratory Disorders |
| Adolescent Medicine & Gynecology | 1 | Adolescent Medicine & Gynecology |
| Sports Medicine & Physical Fitness | 1 | Growth & Development/Preventive Pediatrics; Common Pediatric Disorders; Emergency Pediatric Care |
| Substance Abuse | 1 | Adolescent Medicine & Gynecology; Emergency Pediatric Care |
| Disorders of Cognition, Language & Learning | 1 | Growth & Development/Preventative Pediatrics; Common Pediatric Disorders |
| | 3 | Genetics; Metabolic Disorders; Neurology |
| | 2 | Infectious Disease |
| Psychosocial Issues & Problems | 1 | Growth & Development/Preventive Pediatrics; Common Pediatric Disorders; Adolescent Medicine & Gynecology |
| | 3 | Neurology |
| Critical Care | 1 | Emergency Pediatric Care |
| | 2 | Infectious Disease |
| | 3 | The Fetus & Newborn |
| | 4 | Cardiology; Respiratory Disorders |
| | 5 | Nephrology |
| Emergency Care | 1 | Emergency Pediatric Care; Common Pediatric Disorders |
| Pharmacology | 1 | Emergency Pediatric Care |
| | 2 | Infectious Disease |
| | 4 | Cardiology; Gastroenterology & Nutrition |
| | 5 | Hematology; Rheumatology; Nephrology |
| Research & Statistics | 1 | Growth & Development/Preventive Pediatrics |
| Ethics for Primary Pediatricians | 1 | Common Pediatric Disorders; Adolescent Medicine & Gynecology; Growth & Development/Preventive Pediatrics |
| Patient Safety & Quality Improvement | 1–5 | All Sections |

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## How to Apply for CME Credit

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1. Study the material as often as necessary in order to understand and master the content.
2. Go to www.MedStudy.com and click on CME in the menu bar.
3. Follow the instructions for completing the CME credit application, post-test, and product evaluation.

**Note:** For any questions, please email us at cme@medstudy.com or call 1-800-841-0547, ext. 3.

## Learning Objectives

**As a result of participation in this activity, learners will be able to:**

- Integrate and demonstrate increased overall knowledge of Pediatrics
- Identify and remedy areas of weakness (gaps) in knowledge and clinical competencies
- Describe the clinical manifestations and treatments of diseases encountered in Pediatrics and effectively narrow the differential diagnosis list by utilizing the most appropriate medical studies
- Apply the competence and confidence gained through participation in this activity to both a successful Board exam-taking experience and daily practice

## Target Audience

Participants in this educational activity are those physicians seeking to assess, expand, and reinforce their knowledge and clinical competencies in Pediatrics, focusing their learning on subjects that are directly relevant to clinical scenarios that will be encountered on the ABP Certification or Recertification Board exam, as well as in the practice setting.

## Method of Participation

The content of this CME activity is intended to help learners assess their own key knowledge and clinical competencies with evidence-based standards of care, which are reflected on the Board exams. Pediatricians or other physicians preparing for the ABP certification or recertification (MoC) exam—or who simply want to refresh their knowledge of Pediatrics—should thoroughly read each section of the Core Curriculum two to three times for maximum learning and integration. Pay special attention to yellow-highlighted text, which is considered to be the must-know material for the ABP Board certification and MoC exams, based on ABP exam blueprints. Use Quick Quiz questions to self-assess your learning (answers to these questions are found in the yellow-highlighted text, or in figures and tables). Review tables, figures, and images to reinforce your text reading and to see concise summaries of interrelated facts and clinical examples in key topic areas. Repeat the self-testing process as often as necessary to improve your knowledge and proficiency and ultimately to ensure your mastery of the material. Participants will be required to complete a post-test as part of the requirements for receiving CME credit for this product.

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## For Further Study


*MedStudy Pediatrics Flash Cards*, 2012–2013 Edition, MedStudy Corporation, Colorado Springs, CO, 2012.

*MedStudy 2012 Pediatrics Board-Style Questions & Answers*. MedStudy Corporation, Colorado Springs, CO, 2012.

*Nelson Textbook of Pediatrics*, 19th Edition. Robert M. Kliegman, Bonita F. Stanton, Joseph W. St. Geme, Nina F. Schor, and Richard E. Behrman. W.B. Saunders. Elsevier Science Health Science Division, New York, NY, 2011.

*Rudolph's Pediatrics*, 22nd Edition. Colin D. Rudolph, Abraham M. Rudolph, George Lister, Lewis R. First, and Anne A. Gerson (eds). McGraw Hill Medical, New York, NY, 2011.

*Oski's Pediatrics: Principles and Practice*, 4th Edition. Julia McMillan, Ralph D. Feigin, Catherine D. DeAngelis, and M. Douglas Jones (eds). Lippincott Williams & Wilkins, Philadelphia, PA, 2006.

*Smith's Recognizable Patterns of Human Deformation*, 6th Edition. Kenneth Jones. W.B. Saunders. Elsevier Science Health Science Division, New York, NY, 2006.

*Atlas of Pediatric Physical Diagnosis*, 5th Edition. Basil J. Zitelli and Holly W. Davis. Mosby Elsevier Science Health Science Division, New York, NY, 2007.

*Bright Futures: Guidelines for Health Supervision of Infants, Children, and Adolescents*, 3rd Edition. Joseph F. Hagan, Jr., Judith S. Shaw, and Paula M. Duncan (eds). American Academy of Pediatrics, Philadelphia, PA, 2008.

**Web-based:**
National Guideline Clearinghouse: http://www.guideline.gov/
AAP Clinical Practice Guidelines: http://aappolicy.aappublications.org/practice_guidelines/index.dtl
AAP-Endorsed Clinical Practice Guidelines: http://aappolicy.aappublications.org/endorsed_practice_guidelines/index.dtl
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MedStudy

PEDIATRICS BOARD REVIEW

# CORE CURRICULUM

5th EDITION

Book 1 of 5

Topics in this volume:

**Growth and Development / Preventive Pediatrics**

**Common Pediatric Disorders**

**Emergency Pediatric Care**

**Adolescent Health and Gynecology**

Authored by J. Thomas Cross, Jr., MD, MPH, FAAP with Robert A. Hannaman, MD

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A NOTE ON EDITORIAL STYLE: There is an ongoing debate in medical publishing about whether to use the possessive form that adds "'s" to the names of diseases and disorders, such as Lou Gehrig's disease, Klinefelter's syndrome, and others. We acknowledge there is not a unanimous consensus on this style convention, but we think it is important to be consistent in what style we choose. For this publication, we have dropped the possessive form. The *AMA Manual of Style*, *JAMA*, *Scientific Style and Format* and *Pediatrics* magazine are among the publications now using the non-possessive form. MedStudy will use the non-possessive form in this Core Curriculum when the proper name is followed by a common noun. So you will see phrasing such as "This patient would warrant workup for Crohn disease." Possessive form will be used, however, when an entity is referred to solely by its proper name without a following common noun. An example of this would be "The symptoms are classic for Crohn's."



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PEDIATRICS BOARD REVIEW

PEDS

**CORE CURRICULUM**

5th EDITION

Authored by J. Thomas Cross, Jr., MD, MPH, FAAP
with Robert A. Hannaman, MD

# GROWTH AND DEVELOPMENT
# PREVENTIVE PEDIATRICS

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# Growth and Development / Preventive Pediatrics

## OVERVIEW OF GROWTH

Pediatric health supervision is a cornerstone of pediatric health care and thus has a significant place on the Boards. Continuity of care is based on a developmental framework that recognizes the consistency of growth changes that occur throughout childhood. For the exam, you must know both normal patterns and common variations.

Two periods of rapid growth are observed: infancy and adolescence. The growth of most body tissues and organs parallels this pattern, with these notable exceptions:

- Brain growth is rapid during the first 6 years of life, with minimal change in head size after age 10.
- Lymphoid tissue volume increases rapidly before puberty and then declines steadily until one achieves adult levels.
- Growth of the reproductive organs is slow until puberty.

What determines growth? It involves genetic, environmental, and hormonal factors. Parental size and patterns of growth correspond well to both absolute size and timing of growth spurts. See Table 1-1 [Know] on predicting midparental height; for most children, adult height should be within 5 cm (2 inches) above or below the calculated midparental height. Maximal growth occurs in the spring and summer. We discuss abnormalities and endocrine control of growth in the Endocrinology section.

At each visit, weigh infants and younger children without their clothes, using the same scale at each visit. (Okay, this is what the books say; I know the real world is very different!) Measure length in the recumbent position until the age of 2; thereafter, measure standing height. Measure head size using a tape measure around the greatest circumference of the occipitofrontal area; the standard is to do this 3 times and take the greatest of the 3 measurements.

Do not judge a single measurement of growing points (i.e., weight, height, or head circumference) as the only indicator of a growth problem. Remember: 5% of the population normally will be outside 2 standard deviations (2.5% will be > 95th percentile and 2.5% will be < 5th percentile)! The key is to evaluate the curves over time! Look at the pattern of growth (a single measurement is not as meaningful). Examples:

- If a child is genetically small (both parents are short or small), he will grow parallel to standard curves at or just below the statistically "normal" range of heights and weights. He will have a normal weight for height, a normal skin-fold thickness, and a bone age consistent with chronologic age.
- On the other hand, the child who suffered "an event," such as a prolonged intrauterine hypoxic period leading to intrauterine growth failure, will typically be small for gestational age at birth and, over time, continue to fall further away from population means on all parameters.
- Postnatal onset of a growth problem will present as "falling off" the previously stable growth curves.

**Table 1-1:** Predicting Midparental Height in Children

**Midparental Height for Girls:**

$$\frac{(\text{Father's height} - 13\text{ cm}) + (\text{Mother's height})}{2}$$

**Midparental Height for Boys:**

$$\frac{(\text{Mother's height} + 13\text{ cm}) + (\text{Father's height})}{2}$$

Growth charts (see example in Figure 1-1) designate growth parameters on a scale using appropriate "percentiles for age." Major divisions (the lines) are noted at the 3rd, 5th, 10th, 25th, 50th, 75th, 90th, 95th, and 97th percentiles. A child who crosses major percentile divisions during the first 2 years of life should always raise concern, but this pattern can also be consistent with normal growth variants, and thus, it may be difficult to differentiate from growth failure. For example, a baby's size at birth is greatly influenced by maternal factors (maternal diabetes, etc.). Influence of these factors changes after birth. Growth charts are also available for different ethnic groups (international adoptees) and for certain diagnoses (Down's, Turner's, etc.).

Constitutional Growth Delay: When a child's birth weight and height are initially normal, but drop off proportionately during the first 2 years of life; this may indicate constitutional growth delay (Figure 1-2). Height and weight eventually become parallel to growth curves

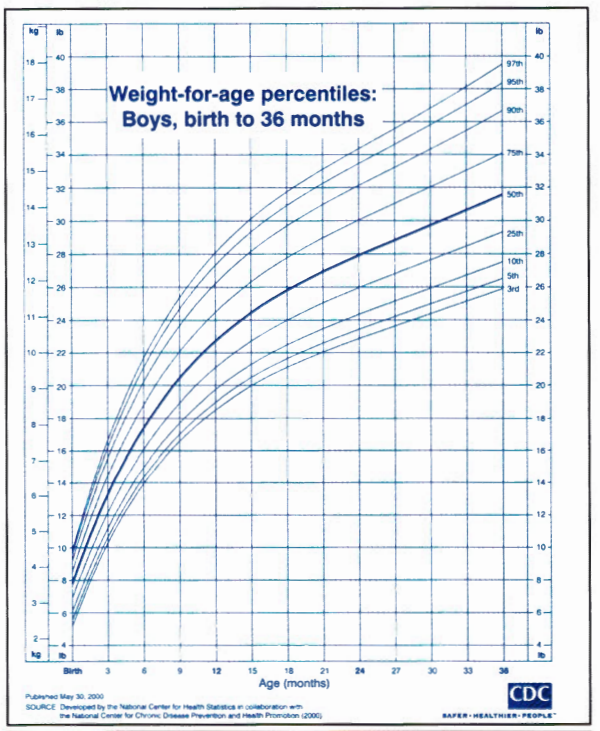



**Figure 1-1:** Sample Growth Chart

at or just below the 3rd percentile for most of middle childhood. Eventually, the older child/adolescent grows rapidly and crosses percentiles to achieve normal adult height. The bone age (BA) is delayed for the child's chronological age (CA) but is consistent with what is known as "height age" (HA, the age at which the child's height is at the 50th percentile). Symbolically you may see this as CA > BA = HA. (Note: In contrast, the bone age is consistent with the chronological age in genetic short stature [Figure 1-3]. CA = BA > HA.) Constitutional growth delays initially may cause concern, but they usually don't manifest as problems until adolescence, and then only if secondary sexual characteristics fail to appear. Also, head circumference is usually relatively spared compared to height and weight. Any significant changes in head circumference should be investigated. Most of the time, constitutional growth delay is hereditary and many parents will report that they too were "late bloomers." "Longitudinal growth charts," which provide normative data for early-, average-, and late-developing children, are helpful in differentiating these kids from those with other growth problems.

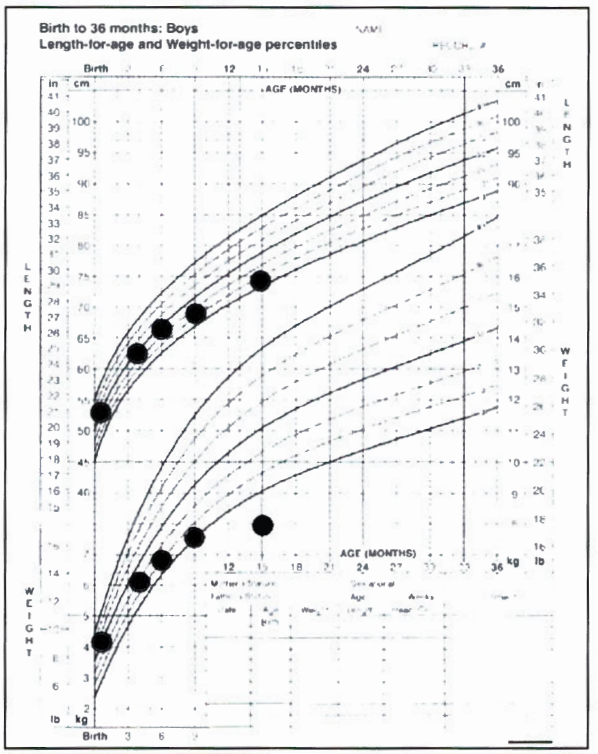



**Figure 1-2:** Plot of Constitutional Growth Delay

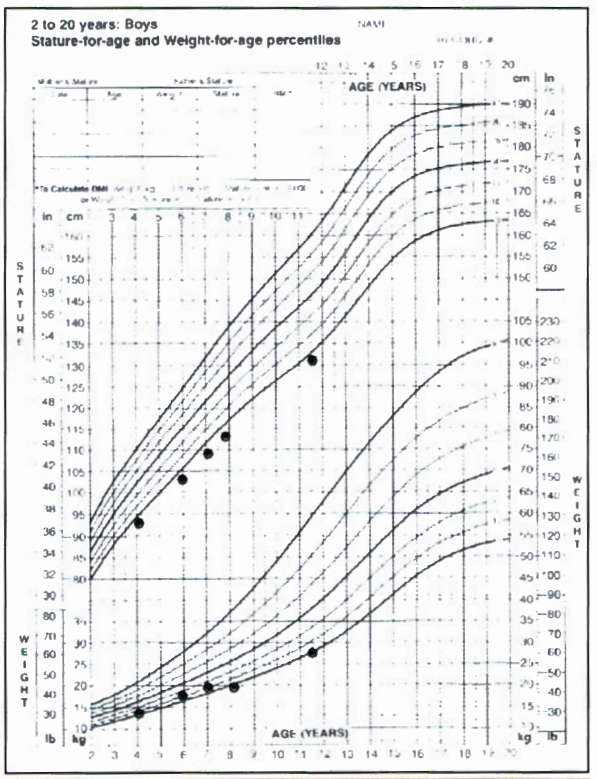



**Figure 1-3:** Plot of Genetic Short Stature

Other quick clues to consider in kids with growth problems:

- Intrauterine insult/genetic abnormality: Weight, height, and head circumference are all significantly below the norms.
- Structural dystrophies/endocrine etiologies: Head circumference frequently will be spared, while height and weight are severely affected. (You can also see this growth pattern in genetic short stature and constitutional growth delay.)
- Caloric insufficiency: Head circumference and height are spared, but significant weight loss is occurring. Over time, though, if caloric insufficiency is persistent and significant, height, and eventually head circumference, will also suffer.

See Table 1-2: Quick Pearls to Remember about Growth for the Board exam. Especially know that birth height doubles by 3–4 years of age. Full-term infants regain their birth weight within 2 weeks, double their birth weight by 4 months, and triple their birth weight by 12 months of age.

Be sure to also know that the 50th percentile for head circumference of a full-term infant at birth is about 35 cm for girls and 36 cm for boys.

## FAILURE TO THRIVE

The term Failure to Thrive, or FTT, is a descriptive term with multiple definitions. It generally applies to weight or changes in weight, but FTT can also include failure to gain length/height and head circumference, depending on the severity and duration of the disorder causing the disruption in growth. See Table 1-3 for common definitions, but note that there is wide variation. Risk factors for

## Quick Quiz

- What is the expected height of a girl, if the father is 182 cm and the mother is 152 cm?
- In constitutional growth delay, is normal adult height achieved?
- How soon does an infant regain birth weight?
- When does birth weight double? Triple? Quadruple?
- When does birth length double? Triple?
- At what percentile is FTT defined?
- True or false? Downward crossing of 3 major percentile divisions would be considered FTT.

FTT include prematurity, congenital malformations, and intrauterine exposures (infections, toxins, drugs, etc.).

Note: For most cases of FTT, you will not find an identifiable organic etiology. Clue for organic etiologies: They rarely present as just FTT! However, psychosocial and behavioral feeding problems are very common, and many texts recommend that these problems no longer be diagnoses of exclusion! Also, because many organic etiologies are reversible, although rare, it is very important to evaluate nonorganic assessments for FTT at the initial evaluation.

What are the key components of evaluation?

- Review of past/present growth data (emphasis especially on pattern of growth)
- History:
  - Dietary history—24-hour recall
    - If formula feeding, be sure to inquire about how formula is being mixed
    - If breastfeeding, is the milk "in"; does mom hear baby swallowing; observe the baby on mom's breast
  - Chronic vomiting and/or diarrhea
  - Abnormal stools (foul-smelling, fatty, contain mucus, "float")
  - Tiring and/or diaphoresis with feeding
  - Constipation/decreased activity
  - Polydipsia/polyuria
- Physical:
  - Abnormal neurological findings
  - Developmental delays
  - Abdominal distention
  - Dysmorphic features
  - Signs of congenital infection
  - Flank masses/organomegaly
  - Wasting of subcutaneous tissue—especially lateral buttocks
  - Anatomic abnormalities—cleft lip/palate, micrognathia
  - Evidence of hypothyroidism (open posterior fontanelle, umbilical hernia)
- Developmental/behavioral assessment
- Feeding observation
- Assessment of child-parent interaction
- Selected initial laboratory studies based on findings above

Studies can include CBC, Lytes, BUN, Cr, U/A +/– Cx, thyroid functions, hepatic enzymes, total protein/albumin, ESR, CRP, stool for fat and culture, karyotype, and bone age (if height is also a problem).

**Table 1-2:** Quick Pearls to Remember about Growth

| |
|---|
| **Weight:** |
| Birth weight is regained by day 10–14 of life |
| Birth weight doubles by 4 months |
| Birth weight triples by 12 months |
| Birth weight quadruples by 24 months |
| After age 2, normal weight gain is 5 lbs/year until adolescence |
| **Length/Height:** |
| Birth length increases by 50% at 1 year |
| Birth length doubles by 4 years |
| Birth length triples by 13 years |
| After age 2, average height increase is 2"/year until adolescence |
| **Head Growth:** |
| Largest rate of growth is between 0 and 2 months (0.5 cm/week) |

**Table 1-3:** Definitions of Failure to Thrive (FTT)

| |
|---|
| **One Point on the Growth Curve:** |
| Weight < 3rd percentile |
| Weight for height < 5th percentile |
| Weight 20% or more below ideal weight for height |
| **A Series of Points on the Growth Curve:** |
| Weight gain < 20 grams/day from 0 to 3 months of age |
| Weight gain < 15 grams/day from 3 to 6 months of age |
| Downward crossing of ≥ 2 major percentiles |

The key question the Boards are likely to ask you: Should you hospitalize the child?

Here are clues to yes:

- Evidence of physical abuse/neglect
- High risk for abuse/neglect:
  - very disturbed parent
  - abnormal child-parent interaction
  - poor parental functioning
  - extremely stressful home environment
- Severe malnutrition
- Medically unstable
- Outpatient management failure
- Need for close observation

All of that said, most cases of FTT are caused by inadequate consumption of appropriate amounts and/or kinds of food, and you can manage most cases on an outpatient basis. These cases are frequently due to psychosocial and/or behavioral problems. If caloric intake appears to be adequate, search for a condition associated with caloric wasting or increased caloric requirements. Don't forget to have the parent explain formula preparation—mistakes are a common cause of infant undernutrition.

Causes of excessive caloric losses:

- Gastrointestinal disorders (e.g., malabsorption)
- Renal disorders (e.g., renal tubular acidosis)

Causes of increased caloric requirements:

- Cardiopulmonary disorders (e.g., CHF)
- Malignancies
- Hyperthyroidism
- Chronic or recurrent infections (e.g., HIV, primary immunodeficiencies)

If, on the Boards, you are asked to describe one of the best ways to identify specific behavioral or interactional problems, the answer is to watch a feeding session. Also ensure adequate calorie intake in the hospital if outpatient management fails. If the child is not gaining weight with appropriate observed nutrition, you must suspect an organic etiology.

If severe psychosocial FTT is present, children may have gaze disturbances—"wary watchfulness" to total avoidance of eye contact and apathetic withdrawal. Infants may resist being held and prefer interactions with inanimate objects. Most will also exhibit developmental delays in the area of language and social behavior.

Note: Infants with short stature or constitutional delay who have a normal weight for length and normal growth velocity do not have FTT!

## HEAD MALFORMATIONS

Micro- and macrocephaly are easily defined: head circumference (HC) 2 standard deviations below or above the mean, respectively. But don't forget that under this definition, approximately 5% of children (2.5% on each end of the spectrum) will fall into this category. (Remember that bully in high school? He probably really did have a fat head!) For preterm infants, plot gestational age rather than chronological age for HC. Also be aware that the catch-up growth of those with microcephaly will exceed their length/weight growth, and they may appear to have macrocephaly or hydrocephalus. The problem is that these premature kids are at increased risk for hydrocephalus. So be careful if the Boards present a premature kid with an enlarged head compared to the rest of his/her body. It could be normal or abnormal. Again, look at the growth pattern. If you are concerned, ultrasound is the diagnostic test of choice for macrocephaly in a premature neonate.

Normally, the head grows 1 cm per month for the first year, with the most rapid growth occurring in the first 6 months. Brain weight doubles by 6 months and triples by 1 year of age. The majority of head growth occurs by 4 years of age.

So what do you do if you suspect abnormal head growth? The usual: Hx, PE, review of previous growth curves. Also review parental and sibling history, and check the child's head circumferences. Look for coexisting factors in the history and exam, such as developmental delay, focal findings, or skin disorders (e.g., the neurocutaneous syndromes—café-au-lait spots with neurofibromatosis Type 1; ash-leaf spots, Shagreen patch with tuberous sclerosis; facial nevus that includes the distribution of the ophthalmic branch of the trigeminal nerve, with Sturge-Weber syndrome). If it is a young infant and you confirm macrocephaly, the best initial diagnostic study is a head ultrasound because hydrocephalus is the most likely diagnosis. For microcephaly, CT or MRI is the best test to help determine an underlying disease process. CT or MRI is better for microcephaly than ultrasound because bone abnormality or lack of brain development are the most likely causes.

Microcephaly: This is usually a result of a primary or secondary defect in brain development.

Remember: Underlying brain growth is what leads to head growth. Normal intelligence can occur, but mental retardation is more likely. Primary microcephaly refers to the presence of a genetic or chromosomal condition in which mass and/or structural brain growth is abnormal.

In secondary microcephaly, infections (both pre- and postnatal—remember that prenatal infections like CMV [periventricular] and toxoplasmosis [throughout cerebral cortex] are often associated with intracranial calcifications), toxins, and CNS injury arrest previously normal brain development. Because brain growth determines

**Quick Quiz**

- Describe the reasons to hospitalize a child with FTT.
- What causes most cases of FTT?
- When is the period of fastest head circumference growth?
- Name 2 benign causes of macrocephaly that occur in normal infants.
- What is the most common cause of plagiocephaly?

skull growth, poor brain growth may result in premature fusion of the cranial bones. Differentiate this from primary craniosynostosis, which presents with both an abnormally shaped skull and palpably thickened suture lines; neither of these is present in conditions where lack of brain growth causes premature fusion of the cranial bones.

The finding of microcephaly at birth does not differentiate primary from secondary causes. However, an infant with a normal-size head at birth with subsequent development of microcephaly strongly suggests a secondary etiology. Perinatal injury to the CNS rarely causes head growth abnormalities before the age of 4–6 months. Usually these growth abnormalities are associated with multiple neurologic abnormalities. Head CT of infants with primary microcephaly is usually normal or shows brain abnormalities consistent with the specific etiology. Secondary causes of microcephaly will frequently show up with abnormal CT scans. Diagnostic testing may be indicated if a child has microcephaly and abnormal development. Testing may include genetic studies if the child appears syndromic, evaluation for congenital infections, or evaluation for metabolic diseases if the infant is hypotonic or acidotic.

Macrocephaly: This is usually due to excess CSF (hydrocephalus), excess brain tissue (macrencephaly or megalencephaly), thickening of the skull, or subdural or epidural bleed. Also, macrocephaly can be a normal variant (see below). Note that macrocephaly rarely presents alone in the face of inborn or acquired disorders! Megalencephaly means having excess brain tissue due to an increased size or number of brain cells. This could be just a normal anatomical variant without problems, but frequently it also occurs with syndromes or metabolic disorders, including many liposomal lipid storage disorders. An infant with anatomic megalencephaly is generally born with a large head, whereas a baby with a metabolic etiology for megalencephaly will have a normal head size at birth, which then enlarges to macrocephaly during infancy. Usually, the metabolic form is associated with seizures, numerous developmental problems, and signs of increased intracranial pressure. Infants with macrocephaly may need referral to a geneticist to be evaluated for other associated abnormalities (e.g., cardiac or eye findings).

A benign cause of macrocephaly is enlargement of the subarachnoid space. This is fairly common in otherwise normal infants. The infant will have a large head (but within normal range) at birth, but the head circumference will subsequently exceed/parallel the 98$^{th}$ percentile. Head CT shows the enlarged subarachnoid space. You also may see normal or minimally increased ventricular size and a widened sulci and sylvian fissure. This is a predominantly male phenomenon and also is frequently seen in the father.

Genetic megalencephaly is another common variant similar to the previous findings, except that the head CT report is normal, which means the underlying brain is normal. Neither condition requires further workup unless neurologic or developmental problems emerge.

Plagiocephaly: This is asymmetric head growth. It can be caused by alterations of internal and external forces that impact skull growth or be due to abnormalities in bone formation. Note that most vaginally delivered infants have head molding that usually resolves during the first few weeks of life. Deformational flattening from lack of changes in head positions is the most common cause of an asymmetric head shape. This resolves without intervention as the head grows.

Note: There has been a large increase in the number of cases of posterior plagiocephaly due to the recommendation that sleeping infants be placed on their backs to reduce SIDS. It is important to differentiate this from asymmetric growth due to premature closure of one or more of the cranial sutures (e.g., craniosynostosis). Remember that most sutures are closed by 12–24 months of age. Most sutures are ossified by 8 years of age, and fusion is complete by early adulthood.

You can predict the head shape based on the sutures involved. It is mainly a cosmetic problem; however, associated ocular, neurologic, and intracranial pressure problems can occur.

If it involves only 1 suture, it is usually isolated and often occurs with a prevalence of 0.1%. 85% are in Caucasian children with M:F ratio of 3:2.

The normal newborn skull is made up of several membranous bones joined by sutures that are intersected by the anterior and posterior fontanelles. The anterior fontanelle is located in the midline above the forehead at the confluence of the coronal and metopic sutures. The posterior fontanelle is located in the midline at the confluence of the lambdoid sutures. The sagittal suture extends along the midline from the anterior to posterior fontanelle; the 2 lambdoid sutures extend downward and laterally from

the posterior fontanelle; the 2 coronal sutures extend laterally from the anterior fontanelle; and the metopic suture originates at the anterior fontanelle and extends along the midline of the forehead (Figure 1-4).

Premature closure of a suture or sutures results in the inability of bone to be laid down along the suture which prevents growth across the suture. As a result, the skull can grow only parallel but not perpendicular to the suture. The most common type of craniosynostosis, premature fusion of the midline sagittal suture, is known as scaphocephaly and results in a long narrow skull. Premature closure of a coronal suture, the second most common type of craniosynostosis, is known as anterior plagiocephaly. It results in asymmetry of the orbits, creating what is often referred to as a "windblown" or "drawn-up" appearance to the orbit on the involved side. The third most common type of craniosynostosis, known as trigonocephaly, is the result of premature closure of the metopic suture, causing a triangle-shaped skull associated with a midline vertical ridge running along the forehead. Posterior plagiocephaly, premature closure of a unilateral lambdoid suture, is uncommon and is easily confused with positional plagiocephaly. Physical examination is very helpful in differentiating positional plagiocephaly from craniosynostosis. Positional plagiocephaly is associated with a parallelogram-shaped head with ipsilateral occipitoparietal flattening, ipsilateral anterior displacement of the ear and ipsilateral frontal bossing while unilateral lambdoid synostosis is associated with trapezoid-shaped head with ipsilateral occipitoparietal flattening, posterior displacement of the ipsilateral ear, and contralateral frontal bossing. Early referral to a neurosurgeon is recommended in any infant suspected to have craniosynostosis.

Several syndromes are associated with craniosynostosis. Crouzon syndrome is most often associated with bilateral closure of the coronal sutures; they have normal IQ and normal hands and feet. Premature closure of multiple sutures associated with syndactyly is seen in Apert syndrome, and those affected have low IQ and syndactyly (mitten hand) of their hands and feet.

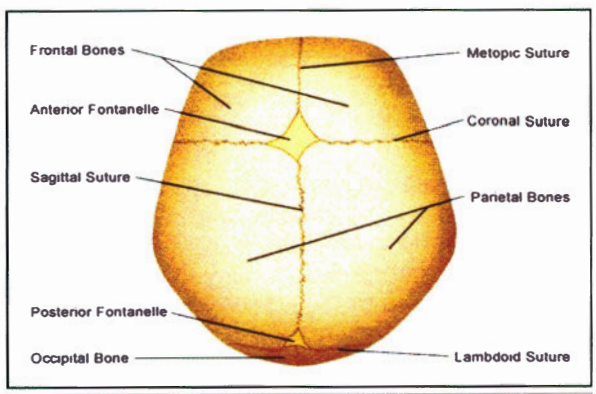



**Figure 1-4:** Normal Newborn Skull

Carpenter syndrome is associated with multiple fusions of the sutures, syndactyly, mental retardation, and, less commonly, congenital heart disease, orthopedic abnormalities, and corneal opacities. Pfeiffer syndrome is associated with brachycephaly or a cloverleaf skull, midface hypoplasia, and finger/toe abnormalities.

Note again: If > 1 suture is involved, assume an association with a genetic disorder and the occurrence of resulting neurologic abnormalities.

Most older texts suggest that if you suspect craniosynostosis, order plain films. If these are inconclusive, a CT scan is the next step. However, many newer references recommend going directly to CT scan with 3D reconstructions, which, in many centers, are now the method of choice for the evaluation of the cranial skeleton and planning of management. The goal of evaluation should not only be the delineation of the various anatomical abnormalities but should also be an attempt at a specific diagnosis. Direct mutation analysis of selected *FGFR* genes may be appropriate in selected cases.

Positional posterior plagiocephaly is usually benign and resolves with time as the child spends less time on his/her back. Encourage the parents to keep the wakeful child in the prone position. Usually the condition will resolve in 2–4 months. For infants with severe or unremitting disease, a helmet may be beneficial (controversial). However, it requires using the helmet 22 hours a day! Helmet molding is most useful when initiated before 6 months of age. Treat synostosis with surgery between 6 and 12 months of age.

## DEVELOPMENTAL MILESTONES

### OVERVIEW

Tip on remembering "Developmental Milestones": Think of your own children and hope they are "normal." (Personally, that's how I got through these questions on the examination.) For these types of questions, I also recommend that, for the next several weeks when you see patients, perform developmental examinations (such as Denver II tool) to reinforce these milestones in your mind. Some of you are visual learners, and Table 1-7 may be helpful to remember milestones. For others of you, it is more useful to break them down by individual milestones; these are listed for you in Table 1-4 through Table 1-11 (Developmental Milestones). Know all of these and try to categorize them in your mind by action/response. Another strategy is to remember a handful of key milestones (two in each category, for example) and extrapolate from there. To reinforce these in your memory, incorporate questions to parents regarding their children's milestones as part of health-maintenance visits or develop an age-appropriate questionnaire for your parents. One problem with developmental milestones is that all sources are not consistent (e.g., some sources say at 30 months a child should make a tower of 8 blocks, while another source says it should be

**Quick Quiz**

- True or false? The premature closure of more than 1 suture is usually associated with other neurologic findings.
- What is the recommended therapy for craniosynostosis?
- Know Table 1-4 through Table 1-11—Developmental Milestones. (Sorry; I know it is a lot.)
- You use the Denver II tool to evaluate a child while he is ill. If he does poorly, should you refer him for further intensive evaluation or repeat the test when he is well?

9 blocks). To add to the confusion, some texts quote these milestones in 1 of 3 ways:

1) On average.
2) 50–90% can do this.
3) > 90% can do this.

To you, the learner (for the exam), this is very disturbing and discouraging; in general, I have gone with what is taught in the 2011 *Nelson Textbook of Pediatrics* 19th edition and in the AAP's Bright Futures.

You absolutely have to know these tables cold. To help you, a few questions that are similar to what may be asked on the test have been added at the end of this section. Again: You absolutely have to know these tables cold, whether you want to or not.

If a parent asks about developmental delay, it is important to take the concerns seriously. Most parents overestimate rather than underestimate their child's skills.

Other common items to remember for the Boards: If a child is ill or uncooperative, consider a "low score" invalid. Chronic disease or recurrent hospitalizations can cause developmental delay. For premature infants, continue age corrections until 18–24 months of age. Physical abnormalities (e.g., hearing, vision, neurologic, or orthopedic), environmental factors (e.g., abuse/neglect), or behavioral problems (e.g., ADHD) may present as a developmental delay. For speech delay, always check hearing first.

A child should be able to copy a circle by 3 years of age. Drawing a cross should occur sometime between 3 and 4. Copying a square won't happen until 4 years of age; a triangle at 5 years of age.

## DEVELOPMENTAL DELAY

Discerning developmental delay can be much more difficult on a written exam without the patient sitting in front of you, so be careful of common clues that the Board exam is likely to give you:

- No head control by 3 months
- Fisting beyond 3–4 months
- Primitive reflexes persisting past 6 months
- Fewer than 50 words/no 2-word phrases by 2 years
- Echolalia beyond 30 months

If you suspect developmental delay, conduct a full history and physical examination. This includes a complete review of systems, prenatal and perinatal history, family history, and psychosocial/behavioral assessment. Guide laboratory testing with history/physical findings.

Generally, the following are recommended:

- For a newborn/infant: Always check any previous metabolic screening done by the state.
- For older children: Serum-lead level, because even low levels can cause problems; also consider thyroid studies (TSH).

**Table 1-4:** Developmental Milestones—Reflexes

| | |
|---|---|
| Moro | Absent by 3–4 months |
| Palmar grasp | Absent by 2–3 months |
| Parachute | Present by 6–9 months |

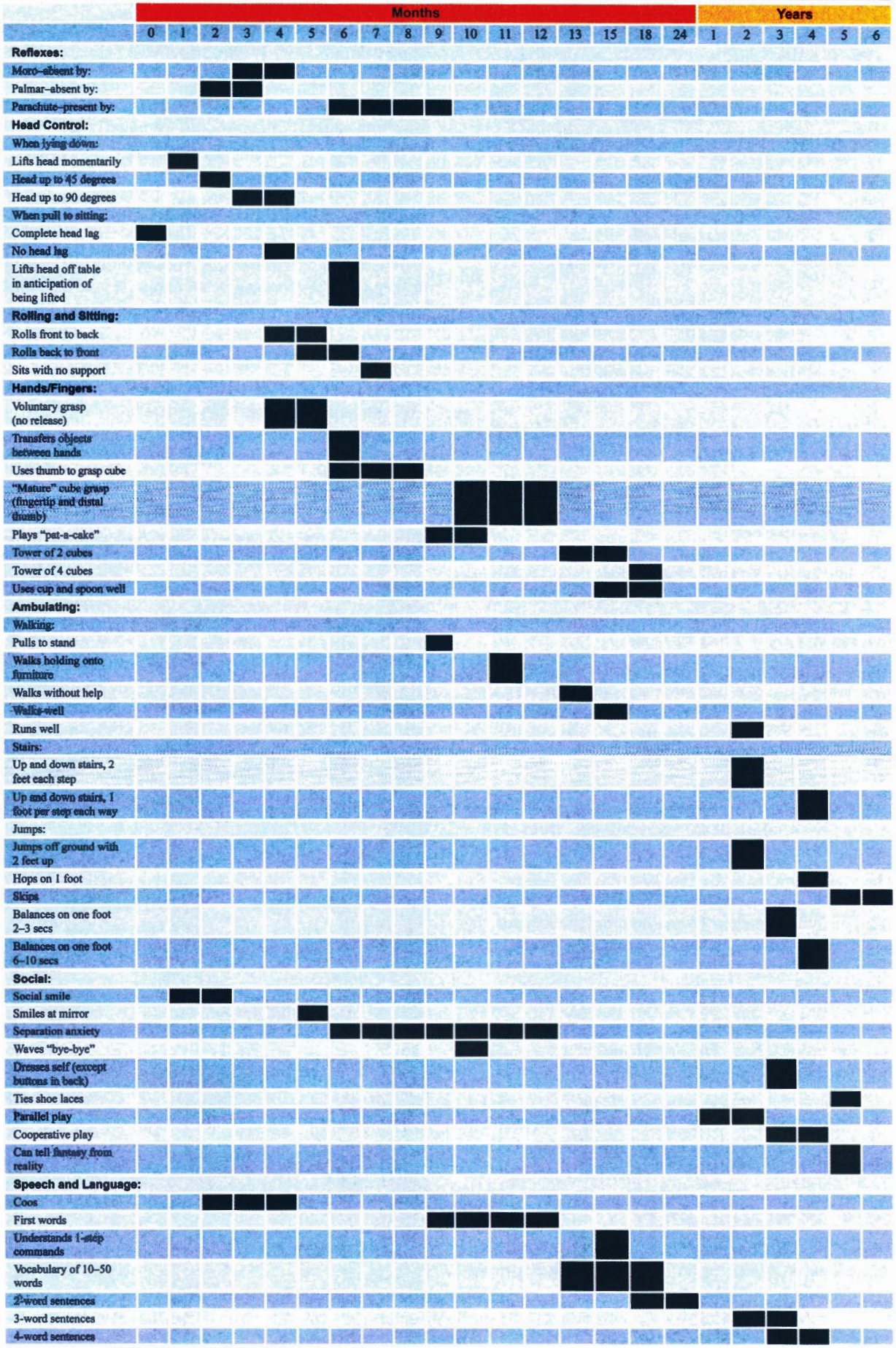
**Table 1-5:** Developmental Milestones—Head Control

| | |
|---|---|
| **When lying down:** | |
| Lifts head momentarily | 1 month |
| Head up to 45 degrees/ can lift head off table | 2 months |
| Head up to 90 degrees/ can lift chest | 4 months |
| **When pull to sitting:** | |
| Complete head lag | Newborn |
| No head lag | 4 months |
| Lifts head off table in anticipation of being lifted | 6 months |

**Table 1-6:** Developmental Milestones—Rolling and Sitting

| | |
|---|---|
| **Rolling:** | |
| Rolls front to back | 4–5 months |
| Rolls back to front | 5–6 months |
| **Sitting:** | |
| Sits with support | 6 months |
| Sits with no support | 7 months |

## Table 1-7: Developmental Milestones



**Quick Quiz**

- True or false? Metabolic screening is required for every child with mental retardation.
- When should you consider ordering metabolic screening?

MRI of the head may detect cerebral dysgenesis at any age in a child. Consider it if any of the following are present:

- Cerebral palsy
- Abnormal head shape or size
- Craniofacial malformation
- Loss or stagnation of developmental skills
- Neurocutaneous abnormalities
- Seizures
- IQ < 50

Metabolic screening is not recommended for asymptomatic children with idiopathic mental retardation.

**Table 1-8:** Developmental Milestones—Hands/Fingers

| | |
|---|---|
| Involuntary grasp | Newborn |
| Grasp reflex disappears, brings hands to midline | 2 months |
| Voluntary grasp (no release) | 4–5 months |
| Raking objects, transfers objects between hands | 6 months |
| Uses thumb to grasp cube | 6–8 months |
| "Mature" cube grasp (fingertip and distal thumb) | 10–12 months |
| Plays "pat-a-cake" | 9–10 months |
| Tower of 2 cubes | 13–15 months |
| Scribbles | 15 months |
| Vertical lines | 18 months |
| Tower of 4 cubes | 18 months |
| Uses cup well | 15–18 months |
| Uses spoon well | 2 years |
| Large buttons | 3 years |
| Fork | 4 years |
| Tie shoes | 6 years |
| Block stacking: | |
| 6 blocks | 24 months |
| 8 blocks | 30 months |
| 3 block bridge | 3 years |
| 5 block gate | 4 years |

Consider metabolic screening if the following signs or symptoms are present with mental retardation:

- Episodic vomiting or lethargy
- Poor feeding
- Poor growth
- Seizures
- Hepatomegaly
- Unusual odors
- Loss of developmental skills
- Sensory abnormality (especially cataracts or retina defects!)
- Acquired skin disorders

Tests for metabolic screening include: fasting plasma amino acids, blood lactate, blood pH and $CO_2$, ammonia, very long-chain fatty acids, urinary oligosaccharides, and urinary mucopolysaccharides.

## MENTAL RETARDATION

Statistically, mental retardation is represented by an IQ of 2 standard deviations below the population mean and is associated with delays in skills, including self-care, social interactions, and communication. This generally corresponds to an IQ of 70–75. "Mild" mental retardation is defined as an IQ of 50–75, and "moderate-to-severe" is an IQ of < 50. More recently, experts recommend basing classification of mental retardation on the child's ability to perform skills of daily living and function, instead of a "number."

**Table 1-9:** Developmental Milestones—Ambulating

| | |
|---|---|
| **Walking:** | |
| Pulls to stand | 9 months |
| Walks holding onto furniture | 11 months |
| Walks without help | 13 months |
| Walks well | 15 months |
| Runs well | 2 years |
| **Stairs:** | |
| Up and down stairs, 2 feet each step | 2 years |
| Up and down stairs, 1 foot per step each way | 4 years |
| **Jumps:** | |
| Jumps off ground with 2 feet up | 2.5 years |
| Hops on 1 foot | 4 years |
| Skips | 5–6 years |
| Balances on one foot 2–3 secs | 3 years |
| Balances on one foot 6–10 secs | 4 years |

"Mild" mental retardation occurs at a rate of 20–30/1,000. It is often hereditary ("you should see his cousin Jesse"), occurs more commonly in low-socioeconomic groups, and is more common in boys. Of note, only 4–8% of those with mild mental retardation have associated identifiable chromosomal abnormalities.

Severe mental retardation occurs at a rate of 1–4/1,000, is sporadic, is more common in boys, and is not associated with socioeconomic factors. 30% of these cases are due to chromosomal abnormalities. Trisomy 21 (Down syndrome) is the most common. History of CNS injury (e.g., from teratogens, infection, and pre-, peri-, or postnatal insults) accounts for 15–20%. Another 10–15% will have cerebral dysgenesis on MRI. You will see multiple congenital anomalies with an identifiable syndrome in only about 4–5% of cases of severe mental retardation. Endocrine/metabolic etiologies account for 3–5%.

If mental retardation is present, do cytogenetic chromosome testing if any of the following is also found:

- Microcephaly
- Family history of mental retardation
- Family history of fetal loss
- IQ < 50
- Skin pigmentary abnormalities
- Suspected genetic syndrome

## SPEECH / LANGUAGE DELAY

50% of children with delayed language development will have delays in other areas.

**Table 1-10:** Developmental Milestones—Social

| | |
|---|---|
| Social smile | 1–2 months |
| Smiles at mirror | 4 months |
| Separation anxiety | 6–12 months |
| Waves "bye-bye" | 10 months |
| Shows or offers toy to adult | 11 months |
| Dresses self (except buttons in back) | 3 years |
| Ties shoe laces | 5 years |
| Symbolic play | 12 months |
| Parallel play, empathy | 24 months |
| Fantasy play | 36 months |
| Cooperative play | 3–4 years |
| Can tell fantasy from reality | 5 years |
| Games with rules | 6 years |

Common causes of language development problems:

- Hearing deficiency (Always order the hearing test first!)
- Mental retardation
- Dysphasia
- Dysarthria
- Structural problems of the mouth/respiratory tract
- Child abuse/neglect

Stages of Normal Speech Development:

- 0–10 months: Early sounds
  - 0–6 months: Localizes sounds, cooing—first at random and then interactively with adults. (Note: Acquisition of simple names such as "mama" and "dada" may occur this early.)
  - 6–10 months: Consonants added to vowels to induce "babbling." (Note: Deaf infants can coo, but don't "interactively" coo or progress to babbling.)
- 10–18 months: "Point and name" period
  - Rapid acquisition of names, especially those beyond "mama" and "dada."
  - 12–18 months: Word-comprehension period Child understands 75–100 words, follows simple

**Table 1-11:** Developmental Milestones—Speech and Language

| | |
|---|---|
| Coos | 2–4 months |
| Squeals | 4 months |
| Babbles | 6 months |
| Mama/dada: non-specifically, polysyllabic babbling | 9 months |
| First words: mama/dada specifically | 9–12 months |
| Understands 1-step commands | 15 months |
| Uses > 5 words; follows simple commands; can identify 4 body parts | 18 months |
| Vocabulary of 10–50 words | 13–18 months |
| 2-word sentences | 18–24 months |
| 100–200 words in vocabulary, speech 50% understood, uses personal pronouns, identifies 6 body parts | 24 months |
| Understands prepositions | 30 months |
| Speaks in 3–4 word sentences, knows hundreds of words, speech is 75% understood, can use plurals, can identify 2 colors, what/who questions | 36 months |
| Speech 100% understood, speaks in paragraphs, uses past/present tense, identifies gender, identifies 5–6 colors, uses "why" questions | 4 years |
| Operational thinking | 6 years |

**QuickQuiz**

- What percentage of children with mild mental retardation have associated chromosomal abnormalities?
- Describe the clinical clues that a child is experiencing language development problems.
- You should be concerned if a child does not speak any words by what age?

commands, and vocalizes "first words." "Pointing" with words is very important at this stage; e.g., pointing and saying "car."

- Older than 18 months: Word-combination period. The ability to combine words usually occurs after being able to speak > 50 words. First sentences are simple constructions usually without prepositions, pronouns, and articles.

Language development is the most widely variable of all of the developmental areas! Children may be labeled as language-delayed when they actually are within acceptable limits. However, it is important to identify a child with genuine language development issues as early as possible!

Clinical evaluation of language skills is quite complicated and usually requires the assistance of an expert.

However, look for these clues to possible language delay problems on the Board exam and in real life:

- Absence of babbling by 9–12 months
- Absence of any words by 18 months
- Absence of meaningful phrases by 24 months
- Speech that is unintelligible (to strangers) by 3 years
- Inability to use language to communicate after 3 years
- Difficulty with language comprehension after 3 years

If you suspect a language disorder, perform a hearing screen (behavioral or brainstem-evoked response audiometry). Again, review the history and exam (including prenatal history and labs), as well as psychosocial factors (abuse/neglect). Once you confirm a language disorder, begin specific treatments.

# STATISTICS

## OVERVIEW

[Know statistics perfectly.]

Screening is one of the most important aspects of a visit to a pediatrician. The value of screening is determined by many factors. You determine a good screening test by its sensitivity and specificity. Because these are so important, we will review these topics now.

## THE BAYESIAN FOURSQUARE

Key: T = true, F = false, P = positive, N = negative

To make sense of the Foursquare used in answering sensitivity and specificity questions, let's go over the

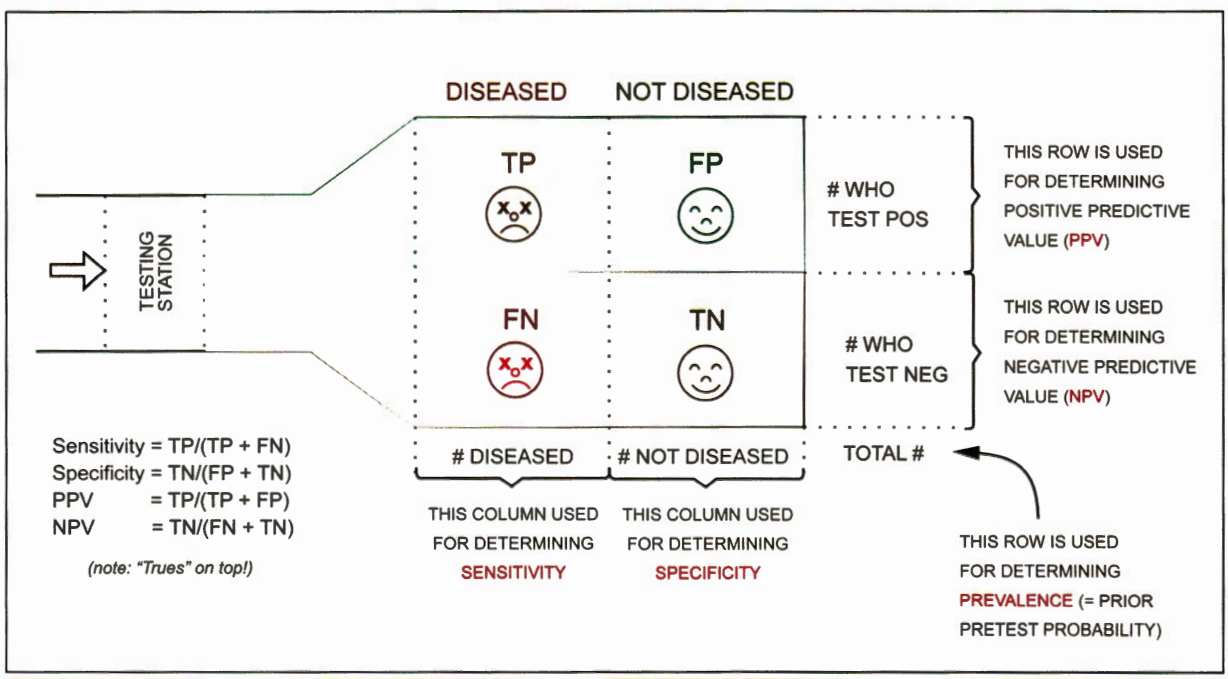



**Figure 1-5:** The Foursquare

diagram in Figure 1-5. Assume we are testing a group of cattle for a deadly disease. These cattle go through the testing station on the left, and you direct them either to the upper corral if their test is positive or to the lower corral if their test is negative. You then drive them all the way across the corral to the right, but this disease is so deadly, all the diseased cattle die before they get to the far right of the corral. So we are left with the four sets of cattle. The upper corral contains those who have the disease and tested positive (true positives = TP) and those who don't have the disease but tested positive (false positives = FP). The lower corral contains those who have the disease and tested negative (false negative = FN) and those who don't have the disease and tested negative (true negatives = TN). The Foursquare is very useful in determining sensitivity, specificity, and positive and negative predictive values.

Sensitivity takes into account only those who have the disease. Sensitivity = true positives (# of patients with disease who test positive) divided by the total # of patients with disease (those who test positive plus the false negatives); thus, sensitivity = TP/(TP+FN). Specificity takes into account only those who do not have the disease. Specificity = true negatives (# of patients without disease who test negative) divided by the total # of patients without the disease (those who test negative plus the false positives); thus, specificity = TN/(TN + FP).

To help you remember: Sensitivity takes into account only those who have the disease, and specificity takes into account only those who do not have the disease. This means that each one is independent of the prevalence of the disease in the selected population! Disease prevalence is just the percentage of the population with the disease. Some like this mnemonic: SPIN—use a SPecific test to rule IN a hypothesis. Note that specific tests have very few false positives. If you get a positive test, you can count on it being a true positive. SNOUT: use a SeNsitive test to rule OUT a hypothesis. Note that sensitive tests have very few false negatives. If you get a negative test, you can count on it being a true negative.

The "positive predictive value" (PPV) of a diagnostic test is the probability of disease in a patient with a positive test—i.e., PPV = P (disease | positive test). To figure this, you take into account the number of both the true positives and the false positives. This combination does reflect prevalence. The formula is PPV = TP/(TP + FP). Makes sense: Divide true positives by all those who test positive. If a disease is rare, even if the sensitivity and specificity are high, the false positives may greatly outnumber the true positives, making the chance much less that a positive test will correlate with actually having the disease. Use this as one of the main factors to determine whether a screening program is useful. Therefore, in contrast to sensitivity and specificity, which are each independent of disease prevalence, both positive and negative predictive values (see next) are influenced by the prevalence of the disease.

The "negative predictive value" (NPV) of a diagnostic test is the probability of not having a disease if the test is negative; i.e., NPV = P (no disease | negative test). Using the Foursquare, the formula is NPV = TN/(TN + FN) (Table 1-12).

The prevalence (or prior/pretest probability) is simply the fraction of the population who has the disease. This is calculated by:

- (Total with disease)/(Total)
  or
- (TP + FN)/([TP + FN] + [FP + TN])

When all of the data are not given in a question that asks you to find sensitivity, specificity, PPV, etc., it is very useful to quickly sketch a Foursquare-based grid like the one shown in Table 1-13. Insert the given values, then calculate for the blank spaces.

**Table 1-12**

| | Disease | No Disease | Total |
|---|---|---|---|
| **Abn tests** | TP | FP | TP + FP |
| **Nor tests** | FN | TN | FN + TN |
| **Total** | TP + FN | FP + TN | |

**Table 1-13**

I. Sketch this first

| | Disease | No Disease | Total |
|---|---|---|---|
| **Abn tests** | | | |
| **Nor tests** | | | |
| **Total** | | | |

**Table 1-14**

II. Based on the given:

| | Disease | No Disease | Total |
|---|---|---|---|
| **Abn tests** | (1) | (3) | 13,000 |
| **Nor tests** | (2) | (4) | 987,000 |
| **Total** | 5,000 | 995,000 | 1,000,000 |

Note that the 5,000 and 995,000 are the denominators in the sensitivity and specificity equations!

**Table 1-15**

III. Fill in the rest

| | Disease | No Disease | Total |
|---|---|---|---|
| **Abn tests** | 4,950 | 8,050 | 13,000 |
| **Nor tests** | 50 | 986,950 | 987,000 |
| **Total** | 5,000 | 995,000 | 1,000,000 |

**QuickQuiz**

- You have invented a test that is 90% sensitive and 95% specific for cystic fibrosis screening. If you tested 100 children with known cystic fibrosis, how many children would the test show as having this disease?
- Define sensitivity, specificity, positive predictive value, and negative predictive value.

In Table 1-14 and Table 1-15, we solve the Foursquare for the scenario in the next paragraph. First, after a bit of calculation, we will fill in the "givens," and then we'll just add or subtract to fill in the other cells of the table. When all the spaces are filled in, we can easily answer just about any statistics question that might be asked. [Know this stuff!]

Scenario: Incidence of asthma is 1/200 in a population. A test has been developed that screens infants at birth for asthma. In a test under consideration, if sensitivity = 99% and the frequency of abnormal tests in the population is 1.3%, what is the ratio of false positives to true positives, and is this a good screening test? To solve, first fill in the Foursquare grid with all the known information.

If the population is not given, assume 1 million. 1/200 incidence = 5,000 total persons with asthma. 0.013 x 1 million = 13,000 total abnormal tests. Just subtract to find the number without asthma (995,000) and the number of normal tests (987,000) (Table 1-14). Then figure out the other blanks in the order shown: 1, 2, 3, and 4. Blank (1) is the only one requiring thought:

Sensitivity = TP/(TP+FN) or 0.99 = TP/5,000

So: TP = 4,950. (Note that the 5,000 and 995,000 are the denominators in the sensitivity and specificity equations.) The others are found by subtraction (Table 1-15).

Once you fill in the entire matrix, you can solve any problem, provided there is enough information. In this example, PPV = TP/(TP + FP) = 38%—not a good percentage for a screening test! If insufficient data are given to solve the problem, it will become apparent when you are unable to fill in all the blanks.

What happens if you change the threshold for normal in a test? If you increase the threshold for what is normal, you will get more negative tests—both true negatives and false negatives. This will decrease the sensitivity and increase the specificity. Why is this? Assume we did this for the previous example. Because the number of people with and without the disease remains the same, the denominators in the sensitivity and specificity equations remain the same. In the sensitivity equation, the numerator decreases (decreased TP due to increased FN), so sensitivity decreases; i.e., fewer of those with the disease are found by the test. In the specificity equation, the numerator increases so specificity increases; i.e., those testing negative are less likely to have the disease.

As a quick trick, think of the threshold for normal in the test as the line in the Foursquare that divides the top from the bottom. As the threshold increases, the line rises, indicating a decreased number of TP and FP and an increased number of FN and TN. As the threshold decreases, the line goes lower, indicating an increased number of positives and decreased number of negatives. Because the denominator stays the same, just see what happens to TP and TN. If TP increases, sensitivity increases. If TN increases, specificity increases. You also will see that anytime the test normals are redefined, sensitivity will increase at the expense of specificity and vice versa.

As disease prevalence/incidence decreases, the number of false positives increases while the number of false negatives decreases. So the ratio of false positives to false negatives increases. This occurs because there is no change in the sensitivity or specificity of the diagnostic test.

## STUDY DESIGNS

There are various study designs that you should know for the ABP exam.

### Cross-Sectional Study

This is the weakest type of study. You look at the presence of the presumed risk factor and presence of the outcome and measure them at the same time in a population. Usually you can't draw many useful conclusions from these studies. These are rarely done in clinical trials.

### Case Control Study

With this type of retrospective study you take subjects and divide them into groups based on presence or absence of the outcome of interest. The study then compares the frequency of risk factors in each group. An example, let's say we want to look at whether drinking coffee during the teen years causes Alzheimer disease. We'll find patients with Alzheimer disease and patients without Alzheimer disease. Then we retrospectively determine if they drank large amounts of coffee in their teen years. We then compare the rate of coffee drinkers in the Alzheimer versus non-Alzheimer group and see if there is a higher percentage of coffee drinkers in the Alzheimer group. This study is also relatively weak because there are so many factors and confounders that could have occurred over the years, and it would be very difficult to absolutely prove your hypothesis that coffee drinking causes Alzheimer. Usually these studies

provide more of an "association" rather than a cause and effect determination.

### Cohort Study

This is generally a prospective study. Here subjects are divided into groups based upon the presence or absence of the presumed risk factor and then followed over a period of time. At the end of the study, the frequency of the outcome is compared. Here we would look at teenagers and divide them into 2 groups of either coffee drinkers or non-coffee drinkers. Then, we would follow them for 40+ years and determine if the coffee drinkers had a higher prevalence of Alzheimer disease. This study is generally quite powerful and is more likely to help you determine a cause and effect, but realize as well that multiple confounders are likely over 40+ years of study. These studies are generally very expensive to do as well as time-intensive and may not show results for years.

### Randomized Control Trials

Here we randomize subjects into groups. One group receives the intervention (patients and researchers are usually blinded, if possible, to whether they receive treatment or placebo), and they are followed forward in time. At the end of the study, the frequency of the outcome is compared. This type of study reduces the effect of unmeasured (confounding) variables that may influence the outcomes of the study. An example here would be to give diabetic patients an ACE inhibitor or placebo, and then follow them over time and see if an ACE inhibitor prevented development of renal insufficiency.

### Meta-analysis

Meta-analysis is the retrospective analysis of many studies concerned with the same topic. There are several methodological flaws and biases involved with these types of analyses, as well as several severe statistical constraints. Compiling studies with differing Type 1 and Type 2 errors is difficult. Also, methods and definitions used to diagnose a disease may differ (e.g., one study of otitis media might require the examiner to include pneumatic otoscopy during the evaluation of the tympanic membrane while another does not). Other areas of difficulty include ages of participants and assumptions of the magnitude of difference expected between the experimental groups.

## *p* VALUE

The $p$ value is a way of expressing a study's statistical significance. The $p$ value is the probability that an effect as large as or larger than the observed effect would occur if in reality there was no true difference between the groups (i.e., the observed outcome was due entirely to chance). Suppose a randomized trial compares 2 drugs and concludes that Drug A is better than Drug B. The smaller the $p$ value, the more confident we can be that Drug A really is better than Drug B and that this is not simply a chance occurrence. Thus, if a study has a $p$ value of 0.05, the likelihood that the results are due to chance is only 1 in 20 (5%; or $p = 0.05$). $p$ values of $< 0.05$—such as 0.01 or 0.001—imply even greater statistical significance. For some arbitrary reason, a $p$ value $\leq 0.05$ is considered statistically significant.

The value $p \leq 0.05$ as statistically significant is probably all you need to know for most questions on $p$ values, but you should know more of the theory. $p$ value is the probability of the result in question occurring, assuming that the distribution of occurrences used for the calculations is correct. Let's do a rough "for instance": Say you normally see 1 case of giardiasis in your office per week. Then one week, you see 4 cases and wonder if there is an epidemic of giardiasis. How often should you see 4 cases a week, if you normally see only 1 case per week?

What you assume is chance variation results in an average incidence of giardiasis of 1 case per week and that the variation in occurrences is "per" a certain distribution. This is generally called the "null" or "chance" hypothesis. The distribution can be plotted from thousands of cases, or we can further assume it follows a standard distribution; i.e., the Poisson distribution. Assuming this is correct, what is the probability of 4 cases occurring in one week? Consult a table that displays various values of the Poisson distribution and read the $p$ value off the table. In the case of giardiasis, you'll see $p = 0.019$.

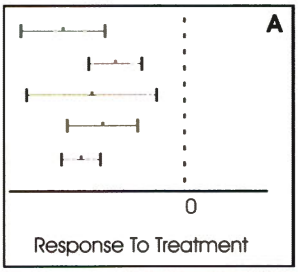



Figure 1-6

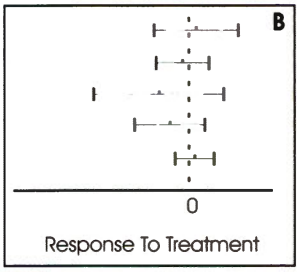



Figure 1-7

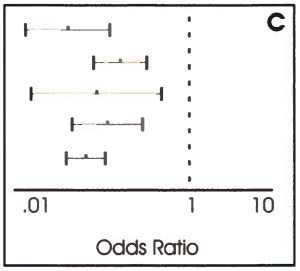



Figure 1-8

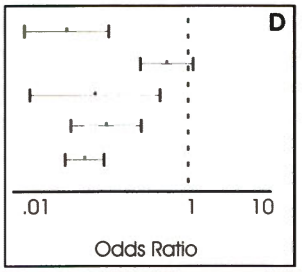



Figure 1-9

**QuickQuiz**

- What *p* value is considered statistically significant?

The way to read this is, "assuming that the average incidence of giardiasis is 1 case per week and further assuming that the incidence of giardiasis follows a normal Poisson distribution, the probability of 4 cases per week occurring by chance is 1.9%." This seems small, and it is, but it does mean that you can expect to see 4 cases per week about once per year (assuming the assumptions are correct). Now, if you see 4 cases again the following week ...!

## TYPE 1 AND TYPE 2 ERRORS

Type 1 = concluding there is a difference (reject null hypothesis) when there is no difference. This is typically expressed by the *p* value. It reflects the willingness of the investigator to declare a benefit when there is none.

Type 2 = concluding there is no difference (fail to reject null hypothesis) when one exists. With Type 2 error, there is the likelihood that the trial will miss a true difference between the two test groups. You can increase the power of the study and decrease Type 2 error by increasing the sample size.

## CONFIDENCE INTERVALS

Confidence interval (CI) charts are frequently used in studies and especially meta-analyses (see below). A CI of 95% is essentially the same as a $p < 0.05$. A CI of 99% is similar to $p < 0.01$. The confidence interval is an estimated range of values that is likely to include an unknown (estimated) population parameter. A 95% CI that does not include the null hypothesis is essentially the same as a $p < 0.05$.

Look at the charts (A–D) in Figure 1-6 through Figure 1-9. The vertical dotted line represents no effect—no response to treatment or an odds ratio of 1. In other charts, the vertical line may be given a specific number that represents the mean from the entire population or from controls. Each horizontal line represents the 95% confidence interval from one study.

If the "95% confidence interval" does not cross the vertical line, then the results are considered significant. For example, if you are reviewing a trial that is looking at response to treatment (vertical line = 0) and see that the 95% confidence interval is 0.5 to 1.9, you know the study shows a significant response (it does not cross the number 0). However, if the 95% confidence interval is –0.7 to 1.6, then that would be a nonsignificant result (because the interval between –0.7 and 1.6 does cross over the number 0)!

These charts (Figure 1-6 through Figure 1-9) may also be shown in a vertical format.

- Chart A is a meta-analysis with each study showing a significant response to treatment.
- Chart B shows a similar meta-analysis in which not even one of the studies shows a significant response to treatment.
- Chart C shows a significantly different odds ratio in all studies reviewed (none of the horizontal lines cross the dashed vertical line). Odds ratio is a way of comparing whether the probability of an event is the same for two groups—usually comparing a control group and the study group (1 = same odds).
- Chart D shows one study with nonsignificant results (the horizontal line crosses the vertical dashed line at 1) but 4 others with significant results; therefore, the meta-analysis will show an overall significant result.

## RELATIVE RISK

Relative risk (RR) measures the strength of association between a risk factor and a disease, and is calculated from prospective cohort studies and randomized controlled trials.

RR = (incidence of disease in those exposed to risk factor) / (incidence of disease in those unexposed to risk factor).

RR > 1 suggests that the risk factor is associated with the disease; i.e., having the risk factor increases the chances the average person would get the disease.

RR < 1 suggests that the risk factor is protective; i.e., having the risk factor gives you less chance of having the disease than the average person would have.

RR = 1 means that the risk factor appears to have association with either risk or protection; i.e., having the risk factor doesn't change your risk from the average person.

95% confidence intervals will use "1" as their magic number to cross over instead of "0" as we saw above.

## ODDS RATIO

Odds ratio (OR) is essentially just an estimate of relative risk (RR) and are used in case control studies. OR has much less power than RR does. Again, the "magic number" for OR will be 1—so you look to see if the 95% confidence interval crosses over 1; if it does, then the study is not significant.

## NUMBER NEEDED TO TREAT

The number needed to treat (NNT) is the number of people who need to be treated for a period of time to prevent one event. NNT is calculated by taking the inverse of the absolute risk reduction between intervention and control groups.

Example: A new drug is studied to see if it can reduce heart failure mortality. Mortality in the treatment arm (active drug) was 10/100, while mortality in the placebo arm was 30/100 during a 4-year follow-up.

NNT = 1/(30/100–10/100) = 1/(0.3 – 0.1) = 1/(0.2) = 5

This means five patients must be treated for 4 years to prevent one death. For good outcomes you generally want NNT to be a relatively small number.

Alternatively, NNH means number needed to harm. It is the number of patients who need to be treated with a drug or intervention to result in one patient being harmed. You generally want this to be a large number.

Not only will you be asked about all of the above, but you will also see sensitivity, specificity, predictive values, and *p* values repeatedly in your medical reading.

# SCREENING IN CHILDREN

## NEWBORN METABOLIC SCREENING

All states (U.S.) currently agree on screening for the following:

- Congenital hypothyroidism
- Hemoglobinopathies (sickle cell, etc.)
- Phenylketonuria (PKU)
- Galactosemia

In addition, tandem mass spectrometry now allows for more than 20 inherited metabolic disorders to be screened with a single test. The average state now screens for between 25 and 30 disorders.

For screening purposes, draw blood before discharge and never later than 7 days of age. PKU requires the "buildup" of metabolites, so if you do a test sooner than 24 hours after birth, repeat it in 1–2 weeks, after buildup of levels becomes high enough to detect but not yet high enough to cause damage. Note: Administration of blood transfusions and dialysis can lead to erroneous results.

## VISION AND HEARING SCREENING

Protocols for vision and hearing are dependent on age and developmental factors, particularly findings that suggest a developmental problem.

### Vision

Here are some general age-based standards and related considerations:

Infancy: By 2 months, infants should track across midline and smile to a smiling face. Object tracking to 180 degrees, red reflex, and conjugate gaze should always be present by 4 months.

Toddler/Preschool: Administer and evaluate unilateral cover test—cover and uncover each eye while the child is looking straight ahead at an object 10 feet away. Movement in the uncovered eye when the opposite is covered or uncovered suggests potential ocular misalignment (strabismus), which requires referral to an ophthalmologist. Refer preschoolers with acuity worse than 20/40 in either eye.

3–5 years of age: Screen with the Random dot E Test. 20% of children will have a refractive error, usually myopia (nearsightedness).

School-aged children, including adolescents: Evaluate these kids annually! Refer children 5–6 years of age who cannot read the majority of the 20/30 line on the vision chart. At all ages, refer a child with a difference of > 1 line in acuity between left and right eyes.

### Hearing

Universal hearing screening is now recommended because deafness is a relatively common finding, with 1–3/1,000 children born deaf. Infants identified with hearing problems before age 6 months are more likely to develop skills similar to their peers entering kindergarten than those identified after 6 months of age. Delayed identification can lead to decreased speech, language, and cognitive abilities compared to peers. Many more children develop sensorineural deficits during childhood. The AAP has consistently recommended universal screening of infants with the goal of 100% screening of all infants by age 3 months.

However, the AAP does not recommend a specific screening modality. Both methods available have problems with sensitivity and specificity. Infants < 6 months have usually been screened with auditory brainstem response testing (ABR). This test measures how CN VIII responds to sound. Clicks or tones are played through soft earphones into the baby's ears. Three electrodes placed on the baby's head measure CN VIII's response. A newer method, known as evoked otoacoustic emissions analysis testing (EOEA), is simpler but has more problems with lower specificity (this means it may incorrectly identify more children with problems, resulting in increased costs due to further unnecessary testing.) This test measures sound waves produced in the inner ear. A tiny probe is placed just inside the baby's ear canal. It measures the response (echo) when clicks or tones are played into the baby's ears. Regardless of which test you choose, the goal is to identify hearing loss of 35 dB or greater in the 500 Hz to 4,000 Hz range. Infants initially screened neonatally should be screened again periodically, depending on the reason for the initial screening; for example, with perinatal CMV, which can result in hearing loss over prolonged periods of time. The AAP additionally recommends formal hearing screening for all children at 3, 4, and 5 years of age and every 2–3 years after that until adolescence.

**QuickQuiz**

- All states (U.S.) screen for which 4 diseases?
- By what age should universal hearing screening be completed?
- List the reasons to screen for hearing loss.
- At what age is blood pressure screening routinely initiated?

Whom should you screen for hearing problems? Screen in all the following situations:

- Parent expresses concerns regarding hearing/language development of a child.
- History of bacterial meningitis.
- Confirmed neonatal infections associated with hearing loss (CMV, HSV, rubella, toxo, syphilis).
- History of significant head trauma (especially temporal bone).
- A syndrome presents that is associated with hearing loss (Treacher-Collins, Waardenburg, osteogenesis imperfecta).
- Exposure to ototoxic medications (gentamicin).
- A neurodegenerative disorder is present (Alport, Cogan).
- Anatomical malformations of the head and neck—especially of the auricle and/or ear canal or if associated preauricular skin tags/dimpling.
- Abnormal pigmentation of the skin, hair, or eyes.
- Heterochromia of the irises.
- Family history of childhood hearing impairment.
- Confirmed incidence of infectious diseases, such as mumps/measles.

## OBESITY / OVERWEIGHT SCREENING

Obesity is a global public health problem, but particularly has expanded (sorry, bad pun) in the U.S. in the last 30–40 years. The National Health and Nutrition Examination Survey from 2005–2008 noted that nearly a third of children older than 2 years of age were classified as "overweight" or "obese" and nearly 17% between 2 and 18 years of age were in the obese range! African-American adolescent girls and Mexican-American boys between the ages of 6 and 12 years had the highest rates of obesity compared to other groups. Higher maternal education seems to confer protection against childhood obesity. Prenatal factors associated with an increased risk of obesity include:

- Weight gain during pregnancy
- High birth weight
- Gestational diabetes

Interestingly, intrauterine growth restriction (IUGR) with early infant catch-up growth is associated with development of central obesity and increased cardiovascular risk.

Body mass index (BMI) is used in children (> 2 years of age) to determine definitions of being overweight or obesity.

BMI is calculated:

$BMI = (\text{weight in kg}) / (\text{height in meters})^2$

Overweight is defined as having a body mass index (BMI) between the 85$^{th}$ and 95$^{th}$ percentiles.

Obesity is defined as having a BMI ≥ 95$^{th}$ percentile.

Treatment of obesity is difficult but a combination of nutritional advice, exercise, and cognitive behavioral approaches is best. Bariatric surgery does work well in adolescents, but the long-term safety has not been established. According to the AAP, "screen time" watching TV or playing video games should be limited to no more than 2 hours/day for children > 2 years of age and children < 2 years should not watch television with the hope that lack of screen time will be replaced with more active behaviors.

## BLOOD PRESSURE SCREENING

Conduct routine blood pressure screening for all children on a yearly basis starting at 3 years of age. Also measure blood pressure in children < 3 years who may have a coexisting medical condition that predisposes them to hypertension (e.g., a toddler who required placement of an umbilical artery catheter following premature birth; an infant with polycystic kidney disease).

Measure BP with the child sitting, with the arm at heart level. Make sure that the width of the cuff bladder is 40% of the circumference of the upper arm at its midpoint, and, when wrapped, the cuff bladder covers 80–100% of the circumference of the arm (thus, the cuff itself must be overlapped significantly). Inflate the cuff to 20 mmHg above the loss of the radial pulse and then deflate at 2–3 mmHg/second. The first sound you hear is a tapping sound, known as Korotkoff sound 1, which is recorded as the systolic blood pressure. The level at which all sounds disappear is Korotkoff sound 5, which is the measured diastolic pressure. Normal BP is defined by a systolic and diastolic pressure < the 90$^{th}$ percentile for age and sex. Blood pressure norms are established for males and females by age and percentile for height. You must determine high-normal (90–95%) and hypertension (> 95%) by 3 separate readings over a period of days to weeks (although depending on the severity of the hypertension and presence of other conditions, you may elect to condense this time frame).

## CHOLESTEROL AND LIPID SCREENING

Universal screening of children for dyslipidemia is not recommended.

The 2008 AAP recommendation is to screen children and adolescents with a positive family history of dyslipidemia or premature (≤ 55 years of age for men and ≤ 65 years of age for women) CVD. It is also recommended that pediatric patients for whom family history is not known or those with other CVD risk factors, such as overweight (BMI ≥ 85$^{th}$ percentile, < 95$^{th}$ percentile), obesity (BMI ≥ 95$^{th}$ percentile), hypertension (blood pressure ≥ 95$^{th}$ percentile), cigarette smoking, or diabetes mellitus, be screened with a fasting lipid profile.

For pediatric patients who are overweight or obese and have a high triglyceride concentration or low HDL concentration, weight management is the primary treatment, which includes improvement of diet with nutritional counseling and increased physical activity to produce improved energy balance. For patients 8 years and older with an LDL concentration of ≥ 190 mg/dL (or ≥ 160 mg/dL with a family history of early heart disease or ≥ 2 additional risk factors present or ≥ 130 mg/dL if diabetes mellitus is present), pharmacologic intervention should be considered. The initial goal is to lower LDL concentration to < 160 mg/dL. However, targets as low as 130 mg/dL or even 110 mg/dL may be warranted when there is a strong family history of CVD, especially with other risk factors, including obesity, diabetes mellitus, the metabolic syndrome, and other high-risk situations. More on screening and treatment in the Cardiology and Metabolic Disorders sections.

## LEAD SCREENING

Lead poisoning remains a significant problem in the U.S.; and thus, the subject appears on the ABP exam! Lead-based paints were banned in the 1970s, but problems still occur from exposure to—and/or ingestion of—lead-containing paint chips, as well as dust that occurs with deterioration and remodeling of older homes. Because it is now recognized that even low lead levels can cause significant clinical effects, the CDC recommends a region-selective screening, based on known prevalence of lead in a particular region, or universal blood lead screening if regional prevalence data are not known, for all children aged 6–72 months. The region-specific measures are for communities where ≥ 12% of 1- and 2-year-olds have blood lead levels ≥ 10 µg/dL, or if ≥ 27% of houses in the area were built before 1950. The interventional threshold has been lowered to levels > 10 µg/dL.

Risk factors to assess beginning at 6 months of age:

- Child lives in, or regularly visits, a house built before 1950.
- Child lives in, or regularly visits, a house built before 1978 that is undergoing remodeling.
- Child has a sibling or play pal who has had an elevated blood lead level.
- The household uses folk remedies that contain lead.
- Child immigrated or was adopted from a country with high lead levels.
- Parental/caretaker exposed to lead via occupation or vocation (e.g., remodeling, pottery with lead paint, working with lead batteries).
- Some urban environments where lead levels are unknown or known to be high.
- Medicaid as the only risk factor is no longer considered to be high risk.

Venous blood sampling is better than capillary (fingerstick) sampling. If a capillary sample is "positive," get a confirmatory venous sample because of the potential for lead contamination on the surface of the skin.

Because lead exposures might change with a child's developmental progress (e.g., walking or reaching window sills) or as a result of external factors (e.g., family relocation or home remodeling), two routine screenings are recommended (at approximately ages 1 and 2 years). Among children in Chicago at high risk with lead levels < 10 µg/dL at age 1 year, 21% had a lead level of ≥ 10 µg/dL when tested again at age ≥ 2 years! Because of this, certain local health departments (e.g., those in Chicago, New York, and Philadelphia) recommend blood lead screening at younger ages or more frequently. For example, these departments recommend lead level testing starting at ages 6–9 months in high-risk areas, blood lead testing at more frequent intervals (every 6 months) for children aged < 2 years, or the provision of additional education and more rapid follow-up blood lead testing for children aged < 12 months with lead levels 6–9 µg/dL.

Lead intoxication may present with numerous nonspecific symptoms that may include abdominal pain with or without vomiting, lethargy and malaise; behavioral changes; and poor school performance. Lead intoxication is associated with a hypochromic microcytic anemia. Basophilic stippling is common but not specific for lead intoxication.

## IRON DEFICIENCY SCREENING

Universal screening for anemia with random hemoglobin/hematocrit is no longer routinely recommended by most organizations; but in their 2008 guidelines, the AAP's Bright Futures guidelines still recommended hematocrit/hemoglobin testing in children 9–12 months of age (and thus for the Board exam).

Screen for the following groups, who are considered to be at increased risk:

- 4-month visit: history of prematurity, low birth weight, use of low-iron formula, and early introduction of cow's milk.

**Quick Quiz**

- Is cholesterol screening universally recommended? If not, for which groups would you consider it?
- At what blood lead level cutoff is intervention recommended?
- True or false? Routine hemoglobin testing is recommended as a screen in 9–12-month-olds (tricky).
- Name children at risk for iron deficiency anemia.
- True or false? Universal screening with urinalysis is no longer recommended by the AAP.
- At what age should you make the initial dental referral?

- 18-month visit; 2-, 3-, 4-, 5-year visits: low-iron diet (low meat diet), poverty, limited access to food, special health care needs.
- 6–10 year visits: Children who consume a strict vegan diet and are not receiving iron supplements.
- 11–21 year visits: All girls should be screened every 5–10 years, with annual screening for those with heavy menstrual flow, low iron intake, eating disorder, or previous diagnosis of iron deficiency.

Do not screen children during an episode of acute illness or for several weeks after fever/infection have abated, because mild transient anemia is common in such circumstances. As in lead screening, venous sampling for iron deficiency is more accurate than capillary sampling. Any abnormal capillary sample must be repeated with a venous sample. Anemia is defined as hemoglobin that is 2 standard deviations below the mean for similar age and sex.

## URINALYSIS SCREENING

Routine screening with urinalysis and urine cultures is not cost-effective. These studies are not recommended by any group, including the U.S. Preventive Services Task Force (USPSTF), AAFP, and the Canadian Task Force on Preventive Health. Finally, the AAP's 2008 Bright Futures Guidelines no longer recommend urinalysis screening! There is good evidence that you should do urine studies only if clinically indicated or if there is concern for renal disease (hereditary factors).

The AAP does recommend annual dipstick urine testing for leukocytes (or other methods to look for *Chlamydia* and gonorrhea) in sexually active males and females between the ages of 11 and 21 years—as part of screening for STDs.

## AUTISM SCREENING

The AAP now recommends universal screening for autism at the 18-month preventative care visit. They recommend an autism-specific tool (e.g., M-CHAT) at the 18-month visit and a repeat specific screening at the 24-month visit—or whenever parental concerns are raised.

The AAP recommends that developmental surveillance be performed at every preventative visit to include eliciting any parental concerns, obtaining a developmental history, making accurate observations of the child, and identifying the presence of risk. A standardized developmental screening tool for children who appear to be at low risk of a developmental disorder should be administered at the 9-, 18-, and 24–30-month visits.

Early signs of autism may include:

- Absence of a social smile at 6 months of age
- No babbling, pointing, or using other gestures by 12 months
- Not using single words by 16 months or 2-word phrases by 24 months
- Loss of language skills or other developmental milestones

Parents often describe their affected infants as "uncuddly," preferring not to be held or swaddled. Other signs to be on the lookout for on the exam include: echolalia, stereotypic or repetitive play, lack of symbolic play, preoccupation with parts of objects, impaired social interactions, limited eye contact, and decreased attention.

## ORAL HEALTH SCREENING

If a child is in one of the following risk groups, she/he should be referred to a dentist as early as 6 months of age and no later than 6 months after the 1st tooth erupts or 12 months of age (whichever comes first):

- Children with special health care needs
- Children whose mothers have lots of caries
- Children with caries, plaque, demineralization, and/or staining
- Children who sleep with a bottle or breastfeed throughout the night
- Children in families of low socioeconomic status

The American Academy of Pediatric Dentistry recommends that all children (even those without risk factors) begin seeing a dentist at 12 months of age (the AAP thinks this is great if a pediatric dentist is available in the community; but if there is not a dentist who is comfortable seeing 12-month-olds, then 3 years of age is recommended as the latest age) and then every 6 months thereafter. Once a tooth erupts, the child's teeth should be brushed twice daily with plain water. Once the child reaches 2 years of age, brush the teeth twice daily with a pea-sized amount of fluoride toothpaste.

# COMMON TOPICS OF WELL-CHILD VISITS

## OVERVIEW

This aspect of health supervision is one of the most important for a pediatrician. However, contemporary time constraints (frequently due to increased overhead, billing, and the need to generate income) have made this particular part of being a pediatrician more difficult than ever. Just remember for the ABP—you always do counseling and guidance at each child visit, no matter what. Be aware that "more" is not always "better." On the Board exam, if you are given options for counseling in a given visit, be sure you "prioritize" the contextual information provided so it relates clinically to the specific reason for the visit; e.g., if the child is in for her 2-month shots, you really don't need to spend much time on the "drugs, sex, and rock and roll" talk (unless this is the child of someone from "the Jersey Shore" MTV show).

In most cases, the rule to follow is basic "common sense." Most counseling revolves around feeding, injury prevention, developmental/behavioral issues, daily care, and medical issues. So, for example, during the 2-month visit, counsel on issues such as:

- Bath safety
- Sun exposure
- Breastfeeding vs. bottle feeding
- Fluoride supplementation, if indicated
- Nutrition topics, such as waiting to introduce solids at 4–6 months
- Sleep, crying, and bowel patterns
- Enjoyment of holding, cuddling, talking to baby (cannot "spoil")
- Immunizations
- Common cold management, including proper use of bulb syringe, saline nose drops, and the dangers of over-the-counter cough and cold preparations
- Child-care arrangements

WHEW!! You are supposed to cover all of that and, oh yeah, examine the baby ... and try to do everything in 15–20 minutes!

An important thing to remember is to be age-appropriate. Notably, the ABP likes to ask about helmets for bicycle riders, initial dental appointment (12 months of age if pediatric dentist available, if not, 3 years at the latest), and, discussion about drugs and sex at age 10 (according to AAP guidelines). Carefully review such age-appropriate subjects as a good Board exam prep.

## NUTRITION ISSUES

### Breastfeeding

Once the mother makes a decision to breastfeed or to feed by bottle, it is important that you are supportive and non-judgmental. Most breastfeeding mothers who wean their infants in the first few weeks postpartum do so because of lack of support or inability to deal with common problems associated with breastfeeding. Questions commonly arise, and it is important that you are available as a resource for the breastfeeding mother. The AAP strongly supports breastfeeding. Data consistently show that breastfeeding provides immediate and long-term benefits, decreasing the incidence or severity of bacterial meningitis, otitis media, diarrhea, and urinary tract infections. Some recent evidence suggests that breastfeeding may offer protection against asthma, diabetes, and obesity. Postnatal infection rates are reduced 21% in breastfed infants, as compared to bottle-fed, in the U.S. Additionally, breastfeeding has been associated with a slight increase in performance on cognitive developmental tests. Breast milk serves as a source of both nutrition and immunologic support: containing immunoglobulins, immune-modulating factors, hormones, growth factors, enzymes, and cholesterol. There are benefits for the nursing mother as well: breastfeeding may reduce the risk of breast and ovarian cancer, hip fractures, and osteoporosis.

Here's a brief physiology review. The newborn sucking on the nipple stimulates the mother's pituitary to release prolactin and oxytocin. These cause the production (remember production and prolactin) and "let-down" (oxytocin) of breast milk. Adequate breast drainage maintains prolactin levels. Medications, maternal fatigue, inadequate fluid intake, and stress can negatively affect these levels. Oxytocin release and the "milk-ejection reflex" occur in response to the infant suckling and increase with rest, warmth, a quiet environment, and the sight of the infant. Pain, embarrassment, distraction, or fatigue can inhibit release. The "milk-ejection reflex" also may cause dripping of milk from the opposite breast while nursing, as well as bring relief of nipple discomfort and uterine cramping. Remember that the newborn is an obligate nose breather—the mother should be reminded to position her baby so that her breast does not cover the baby's nose.

In the first few days of breastfeeding, the baby mainly receives low-volume, antibody-rich colostrum. During this early time, poor feeding or irregular routines rarely impact on future breastfeeding success. However, once milk "has come in" and the mother begins to produce large volumes, poor feeding routines will prevent continued, successful lactation. It is important that mothers do not supplement with a bottle at this point; rather, encourage her to delay the decision for at least several weeks, allowing for a consistent, routine feeding pattern to develop. The infant's motor skills for mouth and tongue movements differ greatly between breastfeeding and bottle feeding. Current guidelines reject rigid schedules for feeding duration and timing. In the initial few days postpartum, most women can attempt

**Quick Quiz**

- At what age should "sex and drugs" be discussed with a child, according to the AAP?
- True or false? It is a good idea to try different formulas to see what helps alleviate "colic."
- What vitamin supplement is recommended for all infants who are exclusively breastfed?
- Which vitamin deficiency is possible in an infant of a mother who is a strict vegetarian?

10–15 minutes per breast with each feed. The infant should take from both breasts at each feeding. Most infants will feed every 2 to 3 hours. Newborns should not go longer than 4–5 hours between feedings. By 2 months of age, most infants will stop one of the middle-of-the-night feedings. The mother should be cautioned against allowing her baby to breastfeed while both she and her baby are lying flat in bed because prolonged suckling while lying flat, and then often falling asleep, may increase the risk of dental caries and accidental suffocation. However, because protein composition of breast milk results in a more rapid digestion time, breastfed infants generally feed more often than bottle-fed infants.

Sore nipples, engorgement, and maternal fatigue are common. Nipple soreness is most frequently due to not using the correct ventral-to-ventral position, inadequate placement of enough of the areola into the mouth to limit the amount of tension on the nipple itself, and/or forgetting to break the suction before removing the baby from the breast. Discourage use of creams and ointments, which will actually exacerbate the soreness. Also encourage the mother not to decrease the number of feeds or duration, because this will lead to engorgement. Inadequate drainage will lead to increased engorgement and involution of the milk supply, which could result in mastitis. If engorgement does occur, increasing the frequency of feedings will alleviate the problem. Use of a pump—or manual milk expression—may also be useful.

The biggest concern generally seems to be whether the baby is "getting enough milk." Remember: Infants may lose 10% of birth weight before regaining it in the first 10–14 days of life. Also, during growth spurts, the baby will decrease the interval between feeds to increase milk production.

## Formula Feeding

Most commercial formulas for healthy full-term infants are cow's milk-based, formed of reconstituted skim milk or skim milk with added whey protein. The carbohydrate source is lactose. The fat consists of a mixture of vegetable oils and removal of the butterfat. All cow's milk–based formulas are essentially the same. Recommend them based on cost and the taste preference of the infant (hmm, okay, it is difficult to ascertain this). Soy-protein formulas today are nutritionally equivalent to cow's milk formulas. Soy formulas are lactose-free and are necessary for those suspected or confirmed of having galactosemia. But note that recent research has found that soy-based formulas have high aluminum levels. There is also evidence that preterm infants do not grow as well on these formulas. For these reasons, do not recommend soy-protein formulas for infants weighing < 1,800 grams. These were once thought to be good "hypoallergenic" formulas, but we now know that there is a high incidence of cross-reactivity between soy protein and cow's milk protein. The fat content in the soy and cow's milk products is essentially the same. One group of infants who may benefit from soy formulas are those with post-diarrheal, transient lactase deficiency. Why? Because the carbohydrate is sucrose and/or corn syrup, and thus it is digested easily in the face of a lactase deficiency—but many nutritional specialists still recommend just "feeding through." Key, however, is to recommend that mothers do not "switch" formulas based on vague GI symptoms or "colic."

Bottle-feeding practices should mimic breastfeeding in that feeding should occur "on demand." This usually means 2–3 ounces every 2–3 hours for newborns with non-feeding intervals lasting no longer than 4–5 hours. After the first week, most infants will take 2–4 ounces every 2–4 hours. By 2 months of age, most bottle-fed infants won't require the "middle-of-the-night" feeding. The 6-month-old child should be taking in < 30 ounces a day, and calories from formula should not exceed 65% of total caloric intake each day. Encourage mothers to avoid giving infants a "go-to-bed" bottle, because this can lead to a significant problem with dental caries. Be sure to counsel against "bottle propping." Most babies can begin weaning from the bottle to cup between 9 and 12 months. Sterilization of bottles and formula is not required. Between uses, bottles simply can be washed with soap and warm water.

## Vitamin and Mineral Supplementation

Vitamin D: In 2008, new AAP guidelines recommended routine vitamin D supplements (400 IU/day) for exclusively and partially breastfed infants in the first few days of life. Supplementation should be continued unless the infant is weaned to at least 1 quart a day of vitamin D-fortified formula or milk. Whole or reduced fat (2%) milk should not be used until after 12 months of age. Levels of vitamin D in breast milk are relatively low, but most used to believe sunlight exposure made up for any deficiency. However, recent reports show an increased risk of rickets in these babies. Breast milk itself is rich in vitamins A and C. Note also that Vitamin $B_{12}$ deficiency has been reported in breastfed infants whose mothers are strict vegetarians. $B_{12}$ and folate deficiency are also common in infants fed goat's milk.

All non-breastfed infants and older children should be given vitamin D supplements if they do not drink at least 1 quart of vitamin D-fortified milk per day. Adolescents who do not obtain 400 IU of vitamin D per day through vitamin D-fortified milk (100 IU per 8 ounce serving) and vitamin D-fortified foods (such as cereals and egg yolks) should receive vitamin D supplementation of 400 IU/day.

Iron: If using formula, always recommend iron-fortified formulations. In 2010, the AAP issued new guidelines for iron supplementation. It is now recommended that full-term, healthy breastfed babies begin daily supplementation with 1 mg/kg of elemental iron at 4 months of age. For infants who are exclusively formula-feeding, provided they are consuming iron-fortified formula, no additional supplementation is recommended. For infants over 4 months of age who are receiving more than 50% of their nutrients from breast milk, the AAP now recommends supplementing with the same amount of elemental iron as the exclusively breastfed infant—1 mg/kg per day. In infants 6 months of age and older, the AAP recommends at least 11 mg of iron a day, preferably from iron-rich foods, and then supplementing with liquid iron as needed. Preterm infants should begin iron supplementation at 2 months of age. You can achieve iron supplementation with infant formula, iron-fortified cereal, or ferrous sulfate drops. Introducing cow's milk prior to 12 months of age can result in occult GI blood loss and worsening of iron deficiency. In addition, iron contained in whole or 2% cow's milk is not readily absorbed, thus adding to the risk of iron deficiency anemia in infants fed whole cow's milk prior to 1 year of age.

Fluoride: This gets a little confusing, depending on the fluoride content of the existing water supply for the household. Note: Do not provide fluoride supplementation to any child under the age of 6 months. If fluoride in the water supply is negligible (< 0.3 PPM), begin supplements at 6 months of age with 0.25 mg, increasing to 0.50 mg at 3–6 years of age, and then to 1 mg at 6–16 years of age. Most bottled water, unless otherwise noted (Nursery® water), does not contain adequate fluoride if mixed with formula or is ingested alone. If the fluoride content is modest (0.3–0.6 PPM), begin supplements at 3 years of age, with 0.25 mg, increasing to 0.5 mg at 6–16 years of age. Any time fluoridation in the water supply is > 0.6 PPM, additional ingestion of fluoride is unnecessary. Excess fluoride can result in fluorosis, a cosmetically disfiguring condition. It is important to counsel parents that children under 6 years of age should use only a "pea-sized" quantity of toothpaste per brushing. Many recommend not using toothpaste until after the age of 2.5 years, because the risk of fluorosis is much higher than the risk of caries in children below this age. If fluoride levels are adequate in the community, and assuming the mother drinks the community water, exclusively breastfed infants will not require fluoride supplements. What about pregnant women living in areas with non-fluoridated water? Latest guidelines recommend not giving fluoride supplementation to pregnant women, because prenatal fluoride supplementation has been shown not to make a difference in the eventual incidence of caries in the offspring.

What about water filters and bottled water? In general, water filters (sink and refrigerator types) used in the U.S. have a negligible effect on the fluoride concentration. Most bottled water, however, does not contain fluoride.

How does fluoride work? Fluoride inhibits dental caries formation by 3 mechanisms:

1) Enhancement of tooth mineralization
2) Reversal of tooth demineralization
3) Inhibition of acid-producing bacteria that cause caries

Fluoride helps calcium and phosphate ions incorporate into enamel, as well as being incorporated itself. Fluoride-containing enamel is much harder and less acid-soluble.

## Advancing the Diet

At 4–6 months of age, most infants are ready to proceed to solid foods. The sequence of beginning such foods is open to debate, and most pediatricians rely on "tradition." This generally means starting with iron-fortified cereals, then strained/pureed vegetables and fruits, followed by meats and poultry products. Most recommend that egg whites, wheat, and fish be introduced later because of concern of GI immaturity and allergic symptomatology commonly associated with these 3 items. Generally, it is best to introduce only one new food at a time and observe for any adverse effect over the next 3–5 days before adding another item.

AAP policy has been to delay whole cow's milk until 12 months of age; and then recommend whole cow's milk instead of skim/2% because of the need for increased dietary fat intake (30–50% of total calories) for optimal growth and development in the first 2 years of life. In 2008, the AAP revised this recommendation and the new consensus is that reduced-fat 2% milk should be given to weaned infants between the ages of 12 months and 2 years if they are at risk of being overweight or have a family history of high cholesterol, obesity, or heart disease.

After age 2 years, all children can gradually be transitioned to 2% and skim milk, generally completing such transition by about age 5.

By 6–9 months of age, most infants can begin using a spoon and cup. Babies usually are competent with these items by 15–18 months. Finger foods become popular by 7–9 months of age. Items to avoid because of the risk of aspiration: raw carrots, large pieces of raw apple, whole or coin-shaped pieces of hot dog, whole grapes, large cookies, peanuts, popcorn, and hard candy.

**QuickQuiz**

- Introduction of cow's milk should be delayed until what age?
- What types of dental trauma should be referred to a dentist?
- What has significantly reduced the incidence of SIDS?

Feeding issues for toddlers: Mealtime routines are very important. Families should sit together and turn the TV off (besides, it seems most shows today have people eating worms or professing their love with a rose—sometimes at the same time). Encourage self-feeding (although it is very messy). Mealtimes should be of finite duration, such as 20–30 minutes. "Cleaning the plate" should not be a sufficient reason for leaving the table. Discourage "grazing" between meals and emphasize the importance of healthy snacks at scheduled periods during the day.

## DENTAL CARE

Primary teeth start to form *in utero,* and permanent teeth start to form soon after birth. At all visits, look for teeth or examine those present, especially at about the 6-month health supervision checkup. First to emerge are the lower anterior teeth (mandibular incisors), followed by the upper opposing teeth (maxillary incisors). Teeth eruption rates and locations can vary significantly, but usually the first tooth erupts by 6 months of age, with 6 teeth present by 1 year (Table 1-16). Eruption cysts (reddish-purple, rounded, raised, fluid-filled lesions) sometimes appear immediately overlying an erupting tooth. Delays in tooth eruption beyond 1 year of age require investigation. While this can be familial, delayed eruption can be associated with hypothyroidism, syndromic disorders, and other developmental conditions.

Toothbrushing should begin with the eruption of the first tooth. Risk of dental caries is increased with more frequent feedings, but the most important factor is the amount of time the child has food in the mouth. Therefore, foods should not be allowed to remain in the mouth at bedtime or naps. "Bottle" caries are very common in children who are allowed to fall asleep with bottles containing milk, flavored drinks, or soft drinks or in children who co-sleep with their mothers and suckle on a breast throughout the night. Usually, the upper anterior and upper/lower posterior teeth are most commonly affected with bottle caries. Additional risk factors for caries include poor parental dental health, prolonged (> 12 months) bottle/breastfeeding, acquired or inherited enamel defects, frequent snacking (including with a sippy cup), crowding/overlapping of teeth, and tooth eruption at < 6 months of age.

**Table 1-16:** Tooth Development

| Teeth | Erupt | Fall out |
|---|---|---|
| Central incisors | 6–12 months | 6–10 years |
| Lateral incisors | 7–16 months | 7–8 years |
| Canines or cuspids | 16–23 months | 9–12 years |
| First molars | 12–19 months | 9–11 years |
| Second molars | 20–33 months | 10–12 years |

Teething is a common concern of parents. Cold items are soothing, and many recommend a frozen pacifier. Acetaminophen or other over-the-counter analgesics can be recommended as well. For the Boards, remember that cases of methemoglobinemia have been reported following the use of topical local anesthetics contained in over-the-counter "teething preparations"!

Dental trauma usually requires evaluation by a dentist, especially if, after a trauma, there is loosening of a secure tooth, chipping or breaking of a crown, laceration of the gingiva, or bleeding around a tooth. If a primary tooth is broken or damaged, it may need to be removed or repaired. Primary ("baby") teeth that are completely knocked out should not be reimplanted. However, permanent teeth should be reimplanted if possible. The avulsed tooth should not be scrubbed or débrided, but rather should be rinsed of visible dirt. If it cannot be reimplanted, it should be kept in Hank's Balanced Salt Solution® (HBSS) or Save-A-Tooth® solution. If these are not available, the next best thing is cold milk, followed by saliva, physiologic saline solution, or any available isotonic solution. Teeth left out longer than one hour have a poor prognosis for reimplantation, but it should be attempted anyway.

Dental infections are best treated with penicillins. If the child is allergic to penicillin, clindamycin is a good choice. Most dental infections are actually due to aerobic bacteria, but anaerobes increase in frequency in those with dental abscesses.

## SLEEP (OR THE LACK OF IT)

The first issue with sleep is the importance of reinforcing the "back to sleep" effort (supine position). Since implementation of this program, the number of SIDS deaths has been reduced markedly. One consequence of "back to sleep" is that children tend to achieve slightly slower early motor milestones (raising their head or chest off the bed when in the prone position), but they "catch up" very quickly once the child is able to roll over to a prone position.

Sleep involves 3 distinct states:

1) Active or REM sleep (rapid eye movements, motor movements, vocalizations, dreaming, and easy awakening).
2) Deeper, quiet or non-REM sleep.
3) In infancy, 50–60% of sleep is spent in REM.

Non-REM intervals last 50–60 minutes. In adults, 25% of sleep is in REM, with intervals of non-REM sleep lasting 90–100 minutes. Newborns sleep 18 hours/day; 6–15-month-olds sleep 10–12 hours at night with 2 nap periods during the day. After 15 months of age, naps decrease to 1/day; by 4 years of age, the naps completely disappear (hmm ... guess I never reached that stage).

Note: It is important for parents to realize that infants will normally wake up at night, and, by 3–4 months of age, they develop the ability to put themselves back to sleep. The problem is usually with the parents, not the child, when they present with a "she just won't sleep all night" complaint. First, parents frequently misinterpret the REM sleep as periods of awakening, and then they attend to the child. In such situations, the parent is actually responsible for the child "waking up." Frequent nighttime feedings with the associated "nocturnal attention" is usually the other culprit. Infants learn nighttime feeding after 6–9 months of age! Infants who always are rocked, fed, or soothed to sleep may be unable to return themselves to sleep without these conditions. Numerous experts recommend that parents allow infants to fall asleep on their own and in their own cribs. This means parents must put the child down awake.

Toddlers are a "whole 'nother can of worms"! Toddlers have an instinctive ability to resist going to bed (that's my philosophy anyway—not proven scientifically). At this age, travel or illness will mess up the best of routines. Normal "separation" issues (i.e., child from parent) also make it more difficult for a 9–18-month-old child to go to sleep. Many recommend the use of a "transition" object, such as a blanket or stuffed toy.

Again, encourage parents to leave their child's room while he/she is still awake, allowing them to settle and fall asleep on their own. Structure in many households is the key for success in this area. Toddlers thrive best on structure, and it is important for them to have a bedtime routine.

## CRYING / COLIC / CRAZY PARENT

Reassure parents that Brazelton did a study showing that a 2-week-old infant cries on average 2 hours/day; this increases to 3 hours/day by 6 weeks and then decreases to 1 hour/day by 3 months. Most crying occurs during the evening hours (but of course; that's when you are tired, just getting home from work, and … well … you get the picture!).

Colic is defined as excessive, unexplained paroxysms of crying in an otherwise well-nourished, normal infant lasting > 3 hours a day and occurring > 3 days a week. 30–50% of kids have colic. The crying usually occurs at the same time during the day (usually those dang evenings) and is resistant to everything you attempt to quell it. During these episodes, the infant may have excess flatus and draw the legs up—making some parents interpret this as "gas" or having "bad belly pains." Usually, it begins in the first week of life and goes away by 3–4 months of age. The cause of colic? No one knows. On the Boards, do not say the answer is to switch formulas if this is the initial presentation for the child with colicky symptoms and there is nothing else to support lactase deficiency!

Methods to improve colicky symptoms? You know the routine here: Each child is different. Some require a quiet environment, some require music, some like to swing at 30 MHz (not 40 or 25 MHz but exactly 30 MHz), some like to ride in a car, and some like to just scream their bloody head off. Whatever the preference might be, there is nothing you can do about it except maybe vacuum your house so you can't hear the crying! On rare occasions, a "colicky" infant may have an identifiable organic cause (corneal abrasion, a hair tied around a digit). Reassure the parents that colic is not the result of something they are doing wrong and be sure the parents of a baby with colic have lots of support and back up.

## DISCIPLINE

This is a difficult one, and we all have our methods, but we will follow the AAP guidelines here, which should mirror the ABP. (Which is why you are reading this, right? You don't need an update on the latest from an afternoon talk show host.) So, for the Boards, corporal punishment is a "No," "NO," and "HECK NO" response. Got it? The ABP likes "time-outs." Parents can use this method as early as 9–12 months and begin to phase it out by 5–6 years of age. Length of time is generally brief—about 1 minute per year of age. (Generally, to a maximum of 5 minutes—WHEW … I told my wife that 50 minutes was too long for me.) If the child leaves the area before time is up, she should be escorted back if she chooses not to return voluntarily. Minimize conversation with the child, and do not discuss the event. After age 5, most children understand loss of privileges, and this is viewed by the ABP as the most effective approach to use at this age. Again, for the purpose of Board questions, never choose corporal punishment as an answer!

## SAFETY AND INJURY PREVENTION

During a health-supervision visit, it is very important to discuss safety and injury prevention. Injuries (both intentional and accidental) are the most common cause of death and morbidity in childhood. 30–75% (depending on age group) of accidental injury-related deaths in children are due to motor vehicle accidents!

Head and facial injuries from bicycle accidents make up a large percentage of emergency department visits and, unfortunately, deaths. Wearing a bicycle helmet is the best way to reduce head and facial injuries. Speaking of bicycle injuries, remember that a youngster who flips his bike and falls on the handlebar may develop a duodenal hematoma—also think of this complication if you see a picture of a quarter-sized bruise on the abdomen or in an abused child with a history of blunt abdominal trauma.

**QuickQuiz**

- True or false? Corporal punishment is viewed as an acceptable means of punishment on the ABP examination.
- What is the safe hot water temperature?

### Child Safety Restraints

Know the various child safety restraints/belts in cars, appropriate for age/height/weight of the child. In 2011, AAP released new recommendations on car safety seats, which included:

Infants and toddlers should ride in a rear-facing car safety seat until they are 2 years of age or until they reach the highest weight or height allowed by their car safety seat's manufacturer.

- Toddlers and preschoolers aged 2 years or older, or those younger than 2 years who have outgrown the rear-facing weight or height limit for their car safety seat, should use a forward-facing car safety seat with a harness for as long as possible, up to the highest weight or height allowed by their car safety seat's manufacturer.
- All school-aged children whose weight or height is above the forward-facing limit for their car safety seat should use a belt-positioning booster seat until the vehicle seat belt fits properly, typically when they have reached 4 feet 9 inches in height and are between 8 and 12 years of age.
- Children who have outgrown their booster seats should ride secured by a lap and shoulder belt in the back seat until 13 years of age.

### Other Items to Review

- Smoke and carbon monoxide detectors
- Cabinet locks and plastic "plugs" for electrical receptacles
- Hot water heater temperature (120° F or less)
- Supervision during bathing
- Animal-bite prevention
- Stair safety
- Poison prevention
- Choking prevention
- Drowning prevention
- Sports safety (rollerblading, skateboarding, football, etc.)
- Firearm safety
- Alcohol and drug abuse
- Suicide prevention
- Homicide prevention

## IMMIGRANT AND INTERNATIONALLY ADOPTED CHILDREN

Immigrant and internationally adopted children are commonly seen by pediatricians today. For non-immigrant children, such as tourists or temporary visitors, no medical evaluation is required upon entry into the U.S. For those children entering the U.S. for a permanent residency visa, the medical examination is limited. For permanent visas, active tuberculosis, HIV, syphilis, gonorrhea, lymphogranuloma venereum, chancroid, and leprosy are supposed to be "excluded." However, for children < 15 years of age, no laboratory testing is required.

Evaluation of the immigrant child depends on several factors. For example, from which country did the child originate? And more specifically, what were the living conditions—was the child living in an orphanage, hospital, or refugee camp?

Common health problems are listed in Table 1-17.

For the examination, be particular about the immunization history. An acceptable immunization record documents the date, dose, and name of the vaccine.

Be on the lookout for these 3 problems:

1) Dose given at too short an interval (e.g., 3-week instead of 4-week minimum)
2) Dose given at too young an age
3) Missing doses

Also be aware that children from orphanages in China or the former Soviet Union may not have produced an adequate immunologic response. If the child is under 1 year of age, the "questionable" doses may just be repeated. For older children, it is generally recom-

**Table 1-17:** Common Health Problems in High-Risk Immigrant Children

| | |
|---|---|
| Infections | Immunization issues<br>Tuberculosis<br>Parasites<br>Hepatitis B<br>Syphilis<br>Malaria |
| Nutritional/Diet | Anemia<br>Malnutrition<br>Rickets<br>Iodine deficiency |
| Toxins | Lead<br>Prenatal alcohol<br>Radioactivity |
| Growth/Development | Estimated age<br>Vision and hearing<br>Dental caries<br>Congenital defects<br>Developmental delay |

mended (and more cost-effective) to determine serum immunity for the major antigens.

As far as screening for infectious diseases, most authorities recommend screening for tuberculosis, intestinal parasites (ova and parasites 3x with specific requests for *Giardia* and *Cryptosporidium*), hepatitis B, congenital syphilis, HIV-1 and -2 infections, regardless of the test results from a foreign laboratory. Tuberculosis is the most common serious infection encountered. Anemia is very common, and children should be retested after treatment of iron deficiency anemia, since correction may unmask other underlying anemias. If the screening CBC shows eosinophilia (absolute eosinophil count > 450 cells) but negative stool ova and parasite examinations, screen for *Strongyloides* with serology. If from endemic areas (sub-Saharan Africa, Southeast Asia, and certain Latin American countries), screen for *Schistosoma* with serology. Rickets is very common in children from China and the former Soviet Union; iodine deficiency occasionally is noted from these countries as well.

Lead poisoning is also common, especially among children from refugee camps and China. Fetal alcohol syndrome rates are very high in children from Eastern Europe and the former Soviet Union.

Vision and hearing screening is also recommended early. As far as "school" issues, most children should be placed in age-appropriate grades.

## "THE SHOTS"

### OVERVIEW

WARNING! KNOW THIS SECTION COLD! There will be several areas you must know really well. These include: the immunization schedule, how to get a child caught up on immunizations, the side effects, the contraindications, which vaccines are live vs. inactivated, and the timing of immunizing with certain products (immunoglobulin, PPD).

### THE SCHEDULE

The latest on this comes out every year! You can find the latest schedule online at http://www.cdc.gov/mmwr. The schedule will usually be in the first or second January issue of the *MMWR*™ for that year. Remember: the ABP is "delayed," so for the October certifying exam, you are probably fine with what is presented in the year of, or even up to 3 years before, your exam. If some new vaccine has come out in the past 6 months to a year before your ABP exam, don't spend a lot of time on it—unless there has been a lot of literature on the vaccine and its trials for several preceding years.

Please see the Immunization Table in the back of this section, taken from the *MMWR* webpage. Also, the schedule is published in the AAP's journal, *Pediatrics*.

By chronological age, then, let's walk through the regimens to be sure you understand them.

Birth: The only vaccine currently recommended at birth is the hepatitis B vaccine. It is mandatory within 12 hours of birth if the mother at the time of delivery is hepatitis B surface antigen-positive (HBsAg+) or the mother's hepatitis B status is unknown. Don't forget to also give the hepatitis B immune globulin (HBIG) immediately to the infant born to the HBsAg+ mother and ASAP (within 1 week, but within 12 hours if the baby is preterm!) to the infant, if the mother turns up being HBsAg+ at the time of delivery room screening.

Although, you can give the shot anytime within the first 2 months when the mother is hepatitis B surface antigen-negative (HBsAg–), the AAP recommends that hospitals have policies and procedures in place that ensure administration of a birth dose of hepatitis B vaccine to all infants ≥ 2 kg at birth unless a physician specifically orders that vaccination be deferred and the mother is confirmed to be HBsAg–. Give the second dose a minimum of 1 month after the first dose. Give the third dose at least 4 months after the first dose and at least 2 months after the second dose, but not before 6 months of age (say that fast 3 times). So, the infant could have any of these regimens for hepatitis B:

- Birth, 1 month, 6 months, or
- 2 months, 4 months, 6 months, or
- 2 months, 4 months, 1 year

Any of these is acceptable, and you don't have to start over if you miss a dose for a year or even several years!

Preterm infants with a birth weight of < 2 kg may have decreased seroconversion rates after administration of hepatitis B vaccine. By one month of chronological age, such infants appear to respond to hepatitis B immunization as do older and larger infants. Therefore, immunization may be delayed in a preterm infant but only if the mother is HBsAg–. Any preterm infant < 2 kg born to an HBsAg+ mother should receive hepatitis B vaccine and HBIG as previously outlined. However, the first dose must not be counted in the required 3-dose series—i.e., a total of 4 doses of vaccine are recommended.

Remember also that infants born to HBsAg+ mothers should be tested after the primary vaccination series for anti-HBs and HBsAg at 9–18 months; nonresponders (anti-HBs negative) who remain HBsAg– should receive an additional 3-dose series at 0, 1, and 6 months.

1 month of age: The only vaccine you could give here is the second dose of hepatitis B—if the infant received the first dose at birth.

2, 4, and 6 months of age: Here is where the fun begins. Now is the appropriate time to give DTaP (D

**QuickQuiz**

- Which vaccine(s) is (are) recommended at birth?

= diphtheria, T = tetanus, aP = acellular pertussis), Hib (*Haemophilus influenzae* type b conjugate vaccine [the bacterial polysaccharide is "conjugated" to protein]), IPV (inactivated polio vaccine), rotavirus vaccine, and PCV (pneumococcal conjugate—here pneumococcal polysaccharides are "conjugated" to nontoxic diphtheria toxin). After this multiple immunization, you give all 5 again at 4 months. At 6 months, you can give all 5 again, or you may elect to not give the IPV—but instead give that one later, anytime between 6 and 18 months. Additionally, at 6 months, if it is the right time of the year, you should administer the influenza vaccine. You also could give the 1st, 2nd, or 3rd hepatitis B vaccine, depending on when/if previous vaccine was given.

Note: All of the vaccines (except oral rotavirus vaccine) that are given before 12 months of age are non-live vaccines (either killed or recombinant).

So, the majority of practitioners give DTaP, Hib, IPV, rotavirus, and PCV at 2, 4, and 6 months of age with 2–3 of the hepatitis B vaccines interspersed in there at your or the parent's discretion. (Note: If using Rotarix® at 2 and 4 months of age, a dose at 6 months is not indicated for this particular rotavirus vaccine. PedvaxHIB® and Comvax®—Hib/HBV—do not require a dose at 6 months of age, while ActHIB® is required at 6 months of age.)

Many individual vaccines are now available in combination products (e.g., DTaP/IPV/HepB—Pediarix®; DTaP/HIB/IPV—Pentacel®) that decrease the number of required shots during the health-maintenance visit.

12 months of age: A final third (or fourth) Hib (depending upon the type of Hib vaccine) and PCV are due sometime between 12 and 15 months of age. The initial MMR (measles, mumps, and rubella) and varicella vaccines are also due at this time. Hepatitis A vaccine is given as well and is universally recommended in the U.S. A second hepatitis A vaccine is recommended 6–12 months after the first. Annual influenza is recommended universally for all children between 6 months and 18 years and for all who have contact with children between these ages.

All children < 9 years of age receiving an influenza vaccine for the first time require administration of a second influenza vaccine ≥ 4 weeks after the first.

15 months of age: The fourth DTaP is due, but you may give the shot as early as 12 months, provided it has been at least 6 months since the third dose.

In summary, by 18 months of age, kids will get:

- 4 DTaP
- 3 or 4 Hib (Note: For PedvaxHIB® or Comvax®, a dose at 6 months is not required.)
- 3 IPV
- 4 PCV
- 1 MMR
- 1 varicella
- 3 hepatitis B
- 2 hepatitis A
- 2 or 3 rotavirus
- 3 influenza (Remember: At 6 months they need 2 shots, each a month apart, for the 1st time they are immunized.)

Also, combination vaccines are being developed to reduce the number of injections an infant/child must receive.

4–6 years of age: When kids get ready for kindergarten, "boosters" for DTaP, IPV, MMR, and varicella are required. However, it is acceptable to give a second MMR as soon as one month after the first MMR and a second varicella as soon as three months after the first varicella vaccine.

11–12 years of age: Kids used to be mostly done with immunizations by this age if they received everything in early childhood. Now, however, we have new vaccines and recommendations! First, a booster Tdap (tetanus, diphtheria, with a new pertussis component) is given at 11–12 years. Human papillomavirus (HPV) is given in a 3-dose series (initial dose, 2 months after the first dose, then 6 months after the first dose) and is recommended at 11–12 years (but can be given as early as 9 years or as late as 26 years). Finally, conjugated meningococcal vaccine (MCV4) is also recommended at 11–12 years of age with a booster dose at 16–17 years of age.

Note: After this Tdap, give a Td booster every 10 years unless a dirty wound has occurred. More on this is in the Infectious Disease section.

Note: The areas shaded under 11–12 years in the immunization table (Figure 2 at the end of this section) recognize that hepatitis B, second MMR, and the 2nd varicella vaccine may not have been either recommended or available when children were initially immunized; so these children need "catching up" at this age.

Also, many females (up to 26 years of age) will require "catch-up" with the HPV vaccine, and many adolescents will require catch-up with MCV4.

I'm hoping that you've been over this a million times by now in your residency, in your practice, and in your sleep. The ABP will ask you multiple questions about when to give vaccines!

## OTHER TIDBITS TO KNOW

All routine childhood immunizations are IM (intramuscular) except for 4 SubQ (subcutaneous)—MMR, varicella, MMRV, and IPV—and 1 oral: rotavirus.

Routinely, only 3 live vaccines are given: rotavirus, MMR (or MMRV), and varicella (oral typhoid and yellow fever vaccines are also live but rarely given, except for travel). Note: It is acceptable to give the live MMR, and in many cases, the varicella vaccine to an HIV-infected person. Note: You can give MMR, varicella, and rotavirus vaccines to a child who lives with an immunocompromised person or whose mother is pregnant!

OPV is contraindicated in both an immunocompromised household and patient.

For IM injections: Use a 20- or 22-gauge 5/8" to 1¼" needle for children; for adults, use a 1½" needle. In infants, use the anterolateral thigh as the injection site; when muscle mass becomes sufficient, you may use the deltoid. Avoid the buttock because of risk of damaging the sciatic nerve and inconsistent intramuscular deposition.

For SubQ injections: Use a 25-gauge 5/8"–3/4" needle for all ages.

Anaphylaxis: If a patient has had an anaphylactic reaction to one of the following, perform skin testing to determine safety of the corresponding vaccine:

- Egg antigens: Influenza, yellow fever
- Streptomycin, neomycin, polymyxin B: IPV
- Neomycin: MMR, varicella
- Gelatin: MMR, varicella, yellow fever

Side effects: With most vaccine administration, some side effects occur—especially local tenderness, mild warmth and erythema, and low-grade fever.

Other key points [Know]:

- A previous anaphylaxis to the vaccine or one of its components: Do not give vaccine.
- Previous mild-to-moderate local reaction to a vaccine (soreness, redness, swelling): Give vaccine.
- Concurrent mild, acute illness with or without low-grade fever: Give vaccine.
- Currently on antibiotics/antivirals: Give vaccine.
- Recent exposure to an infectious disease: Give vaccine.
- History of penicillin or other "nonspecific" allergies: Give vaccine.
- Family history of seizures: Give vaccine.
- Family history of previous adverse reaction to vaccine (particularly DTaP): Give vaccine.
- Active tuberculosis or PPD-positive: Give vaccine after starting TB meds.
- Simultaneous TB skin testing on day of vaccine: Give vaccine. (Note: Measles vaccine may suppress tuberculin reactivity. If you don't give the PPD test on the same day as the MMR, wait 4–6 weeks before placing the PPD.)

Postexposure (within 72 hours) prophylaxis with varicella or MMR vaccine is recommended to prevent or modify disease in susceptible, exposed individuals with no medical contraindications to the vaccine. Note: You can give MMR for postexposure prophylaxis to 6–11-month-olds (before the usual age)—but when they reach appropriate age they should still receive their 2-part MMR series at 12–15 months and 4–6 years. The postexposure prophylaxis dose does not count if the patient is < 12 months!

What about the pregnant adolescent?

The 2009 Red Book states that routine administration during pregnancy is only indicated in the U.S. for Td and inactivated influenza vaccine. However, the AAP does recommend Tdap for pregnant women who have not received a Td-containing booster during the previous 2 years or who are partially immunized. Pneumococcal, meningococcal, hepatitis A, hepatitis B and IPV are considered okay if the patient has an indication or is at risk. Do not give MMR, varicella, intranasal influenza, HPV, or OPV. HPV does not contain live virus but data is too limited in pregnant women. Note: None of these vaccines are contraindicated in children living in households that include a pregnant family member.

What about the breastfeeding mother? All routine vaccines are okay for Mom to receive.

## DTaP / Tdap

DTaP is composed of diphtheria toxoid, tetanus toxoid, and acellular pertussis (contains one or more pertussis antigens). Note: Do not give children > 7 years of age DTaP. Give Tdap (contains a smaller amount of pertussis antigen) for those who are older than 10 years of age (Boostrix®) or 11 years of age (Adacel®). In undervaccinated (catch-up) children, you can also use Tdap in those > 7 years for one dose and then subsequently use Td for the remaining catch-up doses. Prophylactic use of acetaminophen (15 mg/kg) will diminish side effects.

Specifically for DTaP:

- Definitely don't give if [Know these absolutes!]:
  - Hx of encephalopathy within 7 days of dosing.
  - Immediate anaphylactic reaction with previous dose.
  - The patient has a history of a progressive neurologic disorder (you can delay vaccination until the neurologic status is stable or disease process is clarified).
- The following are listed as "probably" don't give again (which for Board purposes means they can't really ask you because it depends on the risk/benefit of the individual child, which you usually can't determine in a quick exam question):

**Quick Quiz**

- Name 3 subcutaneous vaccines.
- Which "live" vaccines are acceptable to give to an asymptomatic HIV-infected child?
- Which vaccine should not be given to an infant if he/she is immunocompromised or if someone in the household is immunocompromised?
- True or false? It is appropriate to give a vaccine to a child with a cold.
- True or false? It is appropriate to give a vaccine to a child with a 101° F fever.
- True or false? A PPD can be placed on the same day that an MMR vaccine is given.
- An MMR was given 1 week ago. A 5-year-old presents for TB skin testing due to possible exposure. Is it appropriate to place the PPD today? If not, how long must you wait?
- Name the vaccines that should not be given to a pregnant adolescent.
- True or false? There is good evidence to support the idea that seizures induced by DTaP cause neurologic damage.
- True or false? An MMR is considered safe in a child with allergy to eggs.
- True or false? If a pregnant woman is inadvertently given MMR vaccine, it is recommended to terminate the pregnancy.

- Hx of fever > 40.5° C (104.8° F), unexplained by another cause, within 48 hours after prior dose
- Collapse or shock-like state (hypotonic-hyporesponsive episode) within 48 hours of previous vaccine
- Seizures within 3 days of receiving prior dose
- Persistent, inconsolable crying lasting > 3 hours within 48 hours
- Guillain-Barré syndrome within 6 weeks of previous tetanus-toxoid-containing vaccine

Note: There is no evidence that seizures after DTaP cause neurologic damage or epilepsy.

## MMR

MMR is a combination vaccine that contains 3 attenuated live viruses (measles, mumps, and rubella). Measles- and mumps-vaccine strains are grown in chick embryos, while rubella is grown in human diploid cell culture. Of interest is that even though measles and mumps vaccines are grown in chick embryos, the vaccines do not contain significant amounts of cross-reacting egg proteins. Therefore, there is no contraindication to vaccinating someone with severe egg allergy with MMR or its components.

Specifically for MMR:

- Definitely don't give:
  - Anaphylactic reaction to neomycin, gelatin
  - Pregnancy (But know that no cases of vaccine-associated congenital rubella syndrome have been reported to date; and if MMR is inadvertently given to a pregnant women, it is not an indication for termination of pregnancy.)
  - Immunodeficiency (Exception: Most HIV-infected children can receive MMR.)
- Probably should not give MMR (which again is not likely to appear on exams):
  - Recent immunoglobulin (blocks antibody response)—try to give at least 2 weeks before giving immunoglobulin, if possible, or wait at least 3–12 months afterward (dose-dependent). (You have to wait 11 months if the patient received IVIG for Kawasaki Disease!)
  - History of thrombocytopenia with the first dose of MMR.
  - History of high-dose (2 mg/kg/day or ≥ 20 mg/day) oral corticosteroid use for 14 days or more—delay at least 1 month from the termination of corticosteroid therapy. (Note that inhaled or topical corticosteroids are not a contraindication to administer MMR.)
- Common adverse events include fever to 103° F, rash occurring 6–12 days after MMR, and joint pain (secondary to the rubella component) 7–21 days after MMR.
- Remember that MMR and PPDs can be given on the same day, but, if you give MMR today, you have to wait at least 4–6 weeks to place a PPD. If you have a measles outbreak at a day care, remember you can give MMR to children as young as 6 months of age for postexposure prophylaxis; otherwise, if younger than 6 months, you'll give immune globulin. Quick question for you … what do you do when a child, exposed to measles at 6 months of age in day care and received MMR, shows up in your office at 12 months of age? That's correct—give her another MMR, and then repeat as usual at 4–6 years of age!

## MMRV

A quick comment on MMRV, which is an option for the 12–15-month and 4–6-year doses. Know that it is associated with more febrile seizures than MMR+V with the 12–15-month dose. The ACIP recommends that practitioners discuss the risks and benefits of MMRV with parents and, unless the parents prefer MMRV, that MMR + V should be given at 12–15 months. MMRV is preferred for the 4–6-year dose. Children with a personal or family history of seizures should not receive MMRV.

Again, note: MMR is safe in those allergic (even anaphylaxis) to eggs or egg products! Note also that

MMR has been proven not to be associated with autism and other neurologic disorders.

## IPV AND OPV

Because of the risk of paralytic disease in both the recipient and contacts, the oral polio vaccine (OPV) is no longer recommended for routine use in the U.S. This is due mainly to the 8 or 9 cases of vaccine-associated paralysis that occurred in the U.S. per year, with most cases occurring in immunocompetent adults. Since IPV is "inactivated," it does not cause paralytic disease. Of note is that the current "enhanced-potency" IPV also does provide some mucosal immunity. The last case of "indigenous" polio appeared in this country in 1979. Since then, more than 125 cases of OPV-associated cases of paralysis occurred—thus, the recommendation is to use IPV instead of OPV in the U.S.

As stated previously, oral polio vaccine, or OPV, is not recommended routinely anywhere in the U.S. However, the Boards still may have an old question about it. Remember, it is a live oral vaccine; so do not give OPV to immunocompromised people—or to children who live in a household with an immunocompromised or (especially on the Boards) an HIV-infected adult (fecal/oral contamination is a concern).

## Hib

*H. influenzae* type b vaccines were first marketed in 1985. Since then, the number of cases of invasive *Haemophilus* infections, including meningitis, has plummeted. The current vaccines are Hib capsular polysaccharides, which are conjugated with various carrier proteins. The use of the "conjugate" markedly enhances the efficacy of the vaccine. However, the vaccine is not effective against the "non-typeable" forms of *Haemophilus* that frequently cause otitis media and upper respiratory infections. For this vaccine, there are no major contraindications.

Note: Do not immunize immunocompetent children > 5 years of age because they are at much lower risk for serious sequelae; and also, they likely have had natural infection by this age. Vaccinate children < 24 months of age who have had invasive *H. influenzae* disease because they may fail to develop immunity following natural infection.

Finally, vaccinate those children with functional/anatomical asplenia (patients with sickle cell, other hemoglobinopathy, or AIDS), regardless of age (even > 5 years).

## HEPATITIS A VACCINE

An inactivated hepatitis A vaccine (Havrix® and Vaqta®) is available. It is given universally at 1 year of age in 2 doses 6 months apart. Virtually all those completing the series develop protective levels of antibody to hepatitis A virus (anti-HAV). Trends based on what is now known of the antibody levels suggest protection for up to 20 years in those who complete the series. The vaccine can also be used for postexposure prophylaxis (for those ≥ 12 months of age) in a household or other exposure setting and is as protective as giving immune globulin.

Even though the HAV vaccine is universally recommended in the U.S., many people may not have received it as a child. Be on the lookout on the Board exam for these scenarios that indicate a person is at high risk for hepatitis A infection or complications:

- High-risk behavior
- Children > 2 years old living in communities with high rates
- Chronic liver disease
- Travel to high-risk countries
- Patients with hepatitis B or C, because these patients can also have fulminant disease if they get hepatitis A

## HEPATITIS B VACCINE

Current hepatitis B vaccines are "recombinant," meaning they use synthetic HBsAg produced in yeast by plasmid gene insertion. The vaccines are now universally recommended for all infants in the U.S. and have been since the early 1990s. Controversy erupted with the discovery that this and several other vaccines contained minute amounts of mercury (thimerosal). This led the AAP and ACIP to recommend that mercury-free vaccines be used in newborns. Both Recombivax HB® and Engerix-B® are now thimerosal-free.

Note: If the mother is HBsAg+ or her status is unknown, give the vaccine at birth or within 12 hours. If mom is HBsAg+, also give HBIG (hepatitis B immune globulin) within 12 hours of delivery at a different injection site. If the mother's status is initially "unknown" and turns out to be positive on testing, give HBIG within 1 week of birth (within 12 hours for preterm!)—but preferably as soon as testing is confirmed positive.

Do not forget to immunize older children and adolescents born after universal early-life immunization began. Be sure you discern if children > 10 years of age were immunized as babies.

## VARICELLA VACCINE

Varicella vaccine was first marketed in the U.S. in 1995. Before its availability, over 10,000 hospitalizations and 100 deaths occurred yearly from chicken pox. Varicella vaccine is made from a live, attenuated virus and contains miniscule amounts of neomycin and gelatin.

For all children, 2 doses are now recommended. The first dose is generally given at 12 months and the second dose at 4–6 years. You can give it with the MMR on the same day; however, if they are not administered simultaneously, give 1 month or more apart.

**Quick Quiz**

- Which vaccines are recommended for patients with sickle cell disease to prevent disease with encapsulated organisms?
- If a mother is known to be hepatitis B surface antigen-positive, what should the infant receive soon after delivery?
- What are the contraindications to receiving varicella vaccine?

Note: 3–5% of children receiving the vaccine will develop a localized varicella-like rash, while another 3–5% will develop a more generalized varicella-like rash within about 1 month after immunization. Although transmission of varicella vaccine virus is very rare, if a vaccine-related rash develops, ensure the child avoids contact with immunocompromised individuals until the rash resolves.

Remember: This is a live-virus vaccine, so do not give to immunocompromised children (except now you may vaccinate children with HIV, hypogammaglobulinemia, and dysgammaglobulinemia). Also, it is okay to give this vaccine to a child who lives with an immunocompromised adult or other child.

You also can safely immunize children with asthma and other conditions when they are on topical, inhaled, or oral steroids in doses < 2 mg/kg/day (or < 20 mg/day for those children over 10 kg).

Do not give the vaccine to pregnant women! But children of pregnant women can receive the vaccine safely.

## PNEUMOCOCCAL CONJUGATE VACCINE

Until February 2000, only a 23-valent polysaccharide pneumococcal vaccine (23PS) was available in the U.S. for prevention of invasive pneumococcal disease. It was mainly given to children with functional/anatomical asplenia, immunodeficiencies, and chronic diseases. In February 2000, a 7-valent pneumococcal conjugate vaccine (PCV7) was released for use for all children < 24 months of age. This vaccine also contains aluminum in small quantities. These 7 serotypes were responsible for 80% of the invasive pneumococcal disease in the U.S. in 2000. The vaccine markedly reduced severe invasive pneumococcal disease and led to a modest but important decrease in acute otitis media, pneumonia, and nasopharyngeal carriage of vaccine serotypes. Overall reduction of invasive pneumococcal disease was 79% (99% decrease in disease caused by serotypes covered in PCV7). The decreases were partially offset by increases in invasive pneumococcal disease caused by non-vaccine serotypes, in particular 19A (which is also associated with increased antibiotic resistance!).

In February 2010, a new 13-valent pneumococcal conjugate vaccine (PCV13) was approved, which includes 19A.

Currently, PCV13 is recommended at 2, 4, 6, and 12–15 months of age. Unvaccinated healthy children aged 24–59 months should receive a single dose of PCV13. Unvaccinated children aged 24–71 months with underlying medical conditions such as asplenia, hemoglobinopathies, cochlear implants, CSF leaks, chronic heart/lung/kidney disease, HIV/ immunodeficiency, DM, malignancy) should receive 2 doses of PCV13 with an interval of at least 8 weeks between doses. A single dose of PCV13 may be administered for children aged 6–18 years who have not received PCV13 previously and are at increased risk for invasive pneumococcal disease because of asplenia, sickle cell disease, immunocompromising conditions such as HIV-infection, cochlear implant, or cerebrospinal fluid leaks, regardless of whether they have previously received PCV7 or PPSV23.

For those children in the "high-risk" groups, give the old 23-valent vaccine after they have finished the 13-valent dosing! The initial dose of the 23PS vaccine should be given at 2 years of age, followed by a repeat dose 5 years later for those with functional or anatomic asplenia or other immunocompromising condition.

## MENINGOCOCCAL VACCINE

There are 2 tetravalent meningococcal polysaccharide-protein conjugate vaccines, MCV4 (Menactra® licensed in 2005, Menveo® licensed in 2010) available for use for persons between 2 and 55 years of age. These vaccines protect against serogroups A, C, Y, and W-135. Remember: 30% of infections are due to serogroup B, so you are still missing a good chunk of infections.

Routine vaccination is recommended for all 11–12-year-olds. In 2011, a booster dose for all 16–17-year-olds was recommended to ensure that patients had adequate protection during the period of highest risk (patients aged 18–23 years and especially college freshmen). Many universities require all college freshmen living in dormitories to receive MCV4, and the military requires that military recruits are immunized. For the Boards, be on the lookout for the word "crowded" to give you a clue to look for meningococcal vaccine.

MCV4 is also recommended for those 2–55 years of age with terminal complement deficiency, properdin deficiency, anatomic or functional asplenia, and certain other high-risk groups. These patients should receive 2 doses 2 months apart for their primary series, followed by a booster dose every 5 years.

HIV patients should receive their first dose of MCV4 at 11–12 years of age, but they need to complete a 2-dose primary series with the second dose 2 months after the first. They should receive a single booster dose at 16–17 years of age.

Individuals 2–55 years of age who are at prolonged risk of exposure (microbiologists and people traveling to endemic areas such as Africa) should receive a single dose of MCV4, followed by a booster dose every 3 years (if 2–6 years of age) or every 5 years (if ≥ 7 years of age).

In October 2005, the Food and Drug Administration (FDA) and Centers for Disease Control and Prevention (CDC) posted an alert about a possible connection between the quadrivalent meningococcal conjugate vaccine and Guillain-Barré syndrome (GBS). Five teens in 4 states developed GBS symptoms 2–4 weeks after receiving MCV4 (Menactra®). Although the rate is similar to that expected without vaccination, the timing is of concern and required further investigation, according to the FDA. The vaccine remains recommended, and since this alert, further increases in Guillain-Barré have not been noted.

MCV4 has replaced the polysaccharide meningococcal vaccine (MPSV4) formerly used for high-risk individuals.

## ROTAVIRUS VACCINE

The rotavirus vaccine first became available in the late 1990s. However, with widespread use, increasing numbers of children developed intussusception, which led to the removal of the vaccine from the market. In 2006, a new vaccine for rotavirus (RotaTeq®) became available and is recommended for administration at 2, 4, and 6 months of age; and, in April 2008, a second vaccine (Rotarix®) was licensed for those at 2 and 4 months of age. If the child is not given the initial rotavirus vaccine by 15 weeks of age, then it is recommended not to give it at all. Also, do not administer the final dose if the child is older than 8 months, 0 days of age, because the vaccine has not been studied in children older than 8 months.

Rotavirus vaccine is a live-attenuated viral vaccine. It is contraindicated in patients with Severe Combined Immunodeficiency, because it can cause prolonged diarrheal disease in these patients.

In U.S. children, the benefits of rotavirus vaccine are substantial. The number of infants and children needing hospitalization or emergency department care for rotavirus disease has decreased by 85%.

In 2010, one study from Mexico found slightly increased intussusception rates associated with the new rotavirus vaccines; however, multiple additional studies (including large U.S. post-marketing studies) show no increased incidence. Considering that the data currently available suggest a possible small risk of intussusception with the rotavirus vaccine but significant benefits in the prevention of rotavirus disease, the CDC continues to recommend rotavirus vaccine to prevent severe rotavirus disease in U.S. infants and children.

However, in late 2011 because of an increased rate of recurrence in those with previous intussusception, the FDA made having a history of intussusception an absolute contraindication to receiving rotavirus vaccine.

## INFLUENZA VACCINE

Inactivated influenza vaccine is recommended universally for all children ≥ 6 months of age. For children < 9 years of age who have never been vaccinated, give 2 doses of vaccine, at least 4 weeks apart, to achieve adequate antibody levels. Thereafter, they receive 1 annual vaccine. If they miss the 2nd dose the first year, they get 2 doses with the next flu season and then go to once annually. Vaccine efficacy is not established for children < 6 months of age.

A live intranasal trivalent flu vaccine (FluMist®) is approved for healthy children > 2 years of age, adolescents, and adults < 50 years of age. It is contraindicated in children < 5 years of age with a history of asthma or recurrent wheezing; in pregnancy; in individuals with a history of severe allergy to chicken eggs; and in individuals with chronic medical conditions that place them at increased risk of complications from influenza infection.

## HUMAN PAPILLOMAVIRUS VACCINE (HPV)

HPV vaccine (HPV4, Gardasil®) was licensed in the U.S. in 2006 and is the first vaccine to prevent cervical cancer, precancerous genital lesions, and genital warts due to human papillomavirus (HPV) types 6, 11, 16, and 18. Types 6 and 11 cause > 90% of all genital warts, while types 16 and 18 account for ~ 70% of all cervical cancers. The vaccine was initially approved for females 9 to 26 years of age and in late 2009 was also approved for males 9 to 26 years of age. HPV is the most common sexually transmitted infection in the U.S. 6.2 million Americans become infected with genital HPV each year, with nearly 10,000 new cases and 3,700 deaths occurring each year due to cervical cancer.

A bivalent HPV vaccine was approved in October 2009 (HPV2, Cervarix®); this vaccine protects against cervical cancer and precancerous genital lesions due to HPV types 16 and 18. This vaccine does not protect against genital warts.

Either HPV vaccine requires a series of 3 IM injections, beginning at age 11–12 years, with the 2nd dose recommended 2 months later, followed by the 3rd dose 6 months after the 1st injection. A history of previous HPV infection is not a contraindication to receiving vaccine.

Syncope can occur after vaccination and has been observed among adolescents and young adults. It is therefore recommended to observe patients for 15 minutes after receiving the vaccine to minimize risk of injury with a syncopal event.

**Quick Quiz**

- Why was the initial rotavirus vaccine in the 1990s pulled from the market?
- A 3-year-old girl receives 1 influenza vaccine but fails to return for follow-up of her next vaccine 4 weeks later. She returns during the next influenza season. How many influenza vaccines does she require this year?
- For whom is HPV vaccine approved?

## EXTRA DEVELOPMENTAL QUESTIONS

1. You are just about to finish a shift in the emergency department when local police officers bring in a little girl they have found wandering in a local city park. The police do not know her name or how old she is. They have brought her to your emergency department to see if you can tell them about how old she is. There is no other history available to you. Her physical examination reveals the following:

   Weight: 11.5 kg
   Length: 82 cm
   Head circumference: 47 cm
   Heart rate: 92
   Respiratory rate: 25
   Temperature: 37.7° C (99.8° F)

   | | |
   |---|---|
   | HEENT: | Clear |
   | Chest: | Clear to auscultation |
   | Heart: | Regular rate and rhythm with no murmur, clicks, or rubs |
   | Abdomen: | Soft with positive bowel sounds, no masses, no hepatosplenomegaly |
   | Extremities: | No cyanosis, clubbing, or edema |
   | Skin: | No rashes |
   | Neuro: | Alert and oriented, cranial nerves 2–12 are grossly intact, all reflexes are 2+/4+ and bilateral, her tone is normal |

   DEVELOPMENTAL: She can run although her gait is somewhat stiff; she can walk up the stairs if you hold her hand; she can make a tower of 4 cubes and imitates scribbling; she can dump a pellet from a bottle. She speaks about 10 words and can identify one or more body parts; she can feed herself but still wears a diaper. She does complain when her diaper is dirty.

   **Which of the following do you tell the police officer, based upon her weight, head circumference, and her developmental examination?**

   A. She is approximately 18 months of age.
   B. She is approximately 24 months of age.
   C. She is approximately 12 months of age.
   D. She is approximately 15 months of age.
   E. You cannot provide them with an approximate age.

2. A mother brings her 9-month-old to see you for his well-child visit. She wants to know if he is on target developmentally.

   **He should be able to do all of the following, except:**

   A. Walk independently
   B. Pull to standing position
   C. Creep or crawl
   D. Use pincer grasp
   E. Use repetitive sounds (mama, dada)

3. A mother brings her 18-month-old in for his well-child exam. This is her first child, and she is very concerned about his development and is always comparing him to other children. You ask her several questions and assure her that he is doing just fine.

   **He should be able to do all of the following, except:**

   A. Identify one or more body parts
   B. Use 2- or 3-word sentences
   C. Scribble
   D. Run stiffly
   E. Feed self

4. A child can copy a circle and a cross for you, but cannot copy a square.

   **Which of the following is her age likely to be?**

   A. 1 to 2 years of age
   B. 5 to 6 years of age
   C. 7 to 8 years of age
   D. 2 to 3 years of age
   E. 3 to 4 years of age

5. **At what age are 90% of children able to sit without support?**

   A. 10 months
   B. 1 year
   C. 5 months
   D. 6 months
   E. 7 months

## ANSWERS

1. **Answer: A.**

**Answer: She is approximately 18 months of age.**

The average weight, height, and head circumference for a 12-month-old girl are approximately 10 kg, 75 cm, and 45 cm. For a 24-month-old girl, the average weight, height, and head circumference are approximately 12 kg, 86 cm, and 48 cm. So reviewing growth curves, her anthropometric values would place her between 12 and 24 months of age. Based upon her developmental examination, she does everything an 18-month-old infant should do. A 12-month-old can walk on level ground with one hand held, but can't do steps; can pick up a pellet with a pincer grasp but not place it in a bottle; and might say only a few words (besides mama, dada). A 15-month-old should walk alone but crawl up stairs, make a tower of 3 cubes and be able to insert a pellet into a bottle, follow simple commands, just start speaking her first real words, and indicate some desires by pointing. A 24-month-old should run well, walk up and down stairs one step at a time, make a tower of 7 cubes, put three words together, and should handle a spoon well.

2. **Answer: A.**

**Answer: Walk independently.**

Most 9-month-olds can do the things listed except for walking independently. Occasionally, you will see a 9-month-old who can walk, but it certainly is of no concern if he doesn't. Usually by 12–15 months, most children can walk.

3. **Answer: B.**

**Answer: Use 2- or 3-word sentences.**

Most children do not begin using 2- or 3-word sentences until they are 24 months old. All of the other answers are abilities typical of 18-month-olds.

4. **Answer: E.**

**Answer: 3 to 4 years of age.**

A child should be able to copy a circle by 3. The cross drawing should occur sometime between 3 and 4. Copying a square won't happen until 4 to 5 years of age. So, if she can get the circle, she has to be older than 3. She can't do the square, which happens between 4 and 5. So she must be less than 4 and more than 3.

5. **Answer: E.**

**Answer: 7 months.**

50% of normal children can sit alone without support by 5½ months, and almost 90% can by 7 months.

Now, practice making your own questions from the developmental areas.

**FIGURE 1. Recommended immunization schedule for persons aged 0 through 6 years — United States, 2011 (for those who fall behind or start late, see the catch-up schedule [Table])**

| Vaccine ▼ Age ▶ | Birth | 1 month | 2 months | 4 months | 6 months | 12 months | 15 months | 18 months | 19–23 months | 2–3 years | 4–6 years |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Hepatitis B[1] | HepB | HepB | | | HepB | | | | | | |
| Rotavirus[2] | | | RV | RV | RV[2] | | | | | | |
| Diphtheria, Tetanus, Pertussis[3] | | | DTaP | DTaP | DTaP | see footnote[3] | DTaP | | | | DTaP |
| *Haemophilus influenzae* type b[4] | | | Hib | Hib | Hib[4] | Hib | | | | | |
| Pneumococcal[5] | | | PCV | PCV | PCV | PCV | | | | PPSV | |
| Inactivated Poliovirus[6] | | | IPV | IPV | IPV | | | | | | IPV |
| Influenza[7] | | | | | Influenza (Yearly) | | | | | | |
| Measles, Mumps, Rubella[8] | | | | | | MMR | | see footnote[8] | | | MMR |
| Varicella[9] | | | | | | Varicella | | see footnote[9] | | | Varicella |
| Hepatitis A[10] | | | | | | HepA (2 doses) | | | | HepA Series | |
| Meningococcal[11] | | | | | | | | | | MCV4 | |

Range of recommended ages for all children

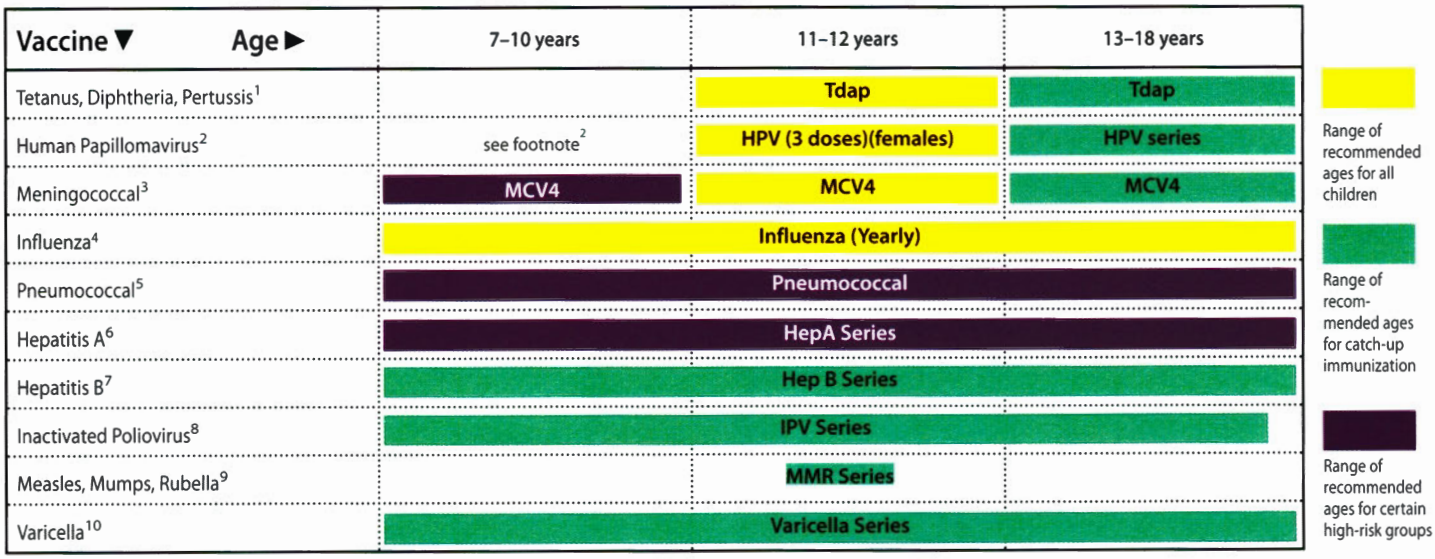
Range of recommended ages for certain high-risk groups

This schedule includes recommendations in effect as of December 21, 2010. Any dose not administered at the recommended age should be administered at a subsequent visit, when indicated and feasible. The use of a combination vaccine generally is preferred over separate injections of its equivalent component vaccines. Considerations should include provider assessment, patient preference, and the potential for adverse events. Providers should consult the relevant Advisory Committee on Immunization Practices statement for detailed recommendations: http://www.cdc.gov/vaccines/pubs/acip-list.htm. Clinically significant adverse events that follow immunization should be reported to the Vaccine Adverse Event Reporting System (VAERS) at http://www.vaers.hhs.gov or by telephone, 800-822-7967.

1. **Hepatitis B vaccine (HepB).** (Minimum age: birth)
   **At birth:**
   - Administer monovalent HepB to all newborns before hospital discharge.
   - If mother is hepatitis B surface antigen (HBsAg)-positive, administer HepB and 0.5 mL of hepatitis B immune globulin (HBIG) within 12 hours of birth.
   - If mother's HBsAg status is unknown, administer HepB within 12 hours of birth. Determine mother's HBsAg status as soon as possible and, if HBsAg-positive, administer HBIG (no later than age 1 week).

   **Doses following the birth dose:**
   - The second dose should be administered at age 1 or 2 months. Monovalent HepB should be used for doses administered before age 6 weeks.
   - Infants born to HBsAg-positive mothers should be tested for HBsAg and antibody to HBsAg 1 to 2 months after completion of at least 3 doses of the HepB series, at age 9 through 18 months (generally at the next well-child visit).
   - Administration of 4 doses of HepB to infants is permissible when a combination vaccine containing HepB is administered after the birth dose.
   - Infants who did not receive a birth dose should receive 3 doses of HepB on a schedule of 0, 1, and 6 months.
   - The final (3rd or 4th) dose in the HepB series should be administered no earlier than age 24 weeks.
2. **Rotavirus vaccine (RV).** (Minimum age: 6 weeks)
   - Administer the first dose at age 6 through 14 weeks (maximum age: 14 weeks 6 days). Vaccination should not be initiated for infants aged 15 weeks 0 days or older.
   - The maximum age for the final dose in the series is 8 months 0 days
   - If Rotarix is administered at ages 2 and 4 months, a dose at 6 months is not indicated.
3. **Diphtheria and tetanus toxoids and acellular pertussis vaccine (DTaP).** (Minimum age: 6 weeks)
   - The fourth dose may be administered as early as age 12 months, provided at least 6 months have elapsed since the third dose.
4. ***Haemophilus influenzae* type b conjugate vaccine (Hib).** (Minimum age: 6 weeks)
   - If PRP-OMP (PedvaxHIB or Comvax [HepB-Hib]) is administered at ages 2 and 4 months, a dose at age 6 months is not indicated.
   - Hiberix should not be used for doses at ages 2, 4, or 6 months for the primary series but can be used as the final dose in children aged 12 months through 4 years.
5. **Pneumococcal vaccine.** (Minimum age: 6 weeks for pneumococcal conjugate vaccine [PCV]; 2 years for pneumococcal polysaccharide vaccine [PPSV])
   - PCV is recommended for all children aged younger than 5 years. Administer 1 dose of PCV to all healthy children aged 24 through 59 months who are not completely vaccinated for their age.
   - A PCV series begun with 7-valent PCV (PCV7) should be completed with 13-valent PCV (PCV13).
   - A single supplemental dose of PCV13 is recommended for all children aged 14 through 59 months who have received an age-appropriate series of PCV7.
   - A single supplemental dose of PCV13 is recommended for all children aged 60 through 71 months with underlying medical conditions who have received an age-appropriate series of PCV7.
   - The supplemental dose of PCV13 should be administered at least 8 weeks after the previous dose of PCV7. See MMWR 2010:59(No. RR-11).
   - Administer PPSV at least 8 weeks after last dose of PCV to children aged 2 years or older with certain underlying medical conditions, including a cochlear implant.
6. **Inactivated poliovirus vaccine (IPV).** (Minimum age: 6 weeks)
   - If 4 or more doses are administered prior to age 4 years an additional dose should be administered at age 4 through 6 years.
   - The final dose in the series should be administered on or after the fourth birthday and at least 6 months following the previous dose.
7. **Influenza vaccine (seasonal).** (Minimum age: 6 months for trivalent inactivated influenza vaccine [TIV]; 2 years for live, attenuated influenza vaccine [LAIV])
   - For healthy children aged 2 years and older (i.e., those who do not have underlying medical conditions that predispose them to influenza complications), either LAIV or TIV may be used, except LAIV should not be given to children aged 2 through 4 years who have had wheezing in the past 12 months.
   - Administer 2 doses (separated by at least 4 weeks) to children aged 6 months through 8 years who are receiving seasonal influenza vaccine for the first time or who were vaccinated for the first time during the previous influenza season but only received 1 dose.
   - Children aged 6 months through 8 years who received no doses of monovalent 2009 H1N1 vaccine should receive 2 doses of 2010–2011 seasonal influenza vaccine. See MMWR 2010;59(No. RR-8):33–34.
8. **Measles, mumps, and rubella vaccine (MMR).** (Minimum age: 12 months)
   - The second dose may be administered before age 4 years, provided at least 4 weeks have elapsed since the first dose.
9. **Varicella vaccine.** (Minimum age: 12 months)
   - The second dose may be administered before age 4 years, provided at least 3 months have elapsed since the first dose.
   - For children aged 12 months through 12 years the recommended minimum interval between doses is 3 months. However, if the second dose was administered at least 4 weeks after the first dose, it can be accepted as valid.
10. **Hepatitis A vaccine (HepA).** (Minimum age: 12 months)
    - Administer 2 doses at least 6 months apart.
    - HepA is recommended for children aged older than 23 months who live in areas where vaccination programs target older children, who are at increased risk for infection, or for whom immunity against hepatitis A is desired.
11. **Meningococcal conjugate vaccine, quadrivalent (MCV4).** (Minimum age: 2 years)
    - Administer 2 doses of MCV4 at least 8 weeks apart to children aged 2 through 10 years with persistent complement component deficiency and anatomic or functional asplenia, and 1 dose every 5 years thereafter.
    - Persons with human immunodeficiency virus (HIV) infection who are vaccinated with MCV4 should receive 2 doses at least 8 weeks apart.
    - Administer 1 dose of MCV4 to children aged 2 through 10 years who travel to countries with highly endemic or epidemic disease and during outbreaks caused by a vaccine serogroup.
    - Administer MCV4 to children at continued risk for meningococcal disease who were previously vaccinated with MCV4 or meningococcal polysaccharide vaccine after 3 years if the first dose was administered at age 2 through 6 years.

**FIGURE 2. Recommended immunization schedule for persons aged 7 through 18 years — United States, 2011 (for those who fall behind or start late, see the schedule below and the catch-up schedule [Table])**




This schedule includes recommendations in effect as of December 21, 2010. Any dose not administered at the recommended age should be administered at a subsequent visit, when indicated and feasible. The use of a combination vaccine generally is preferred over separate injections of its equivalent component vaccines. Considerations should include provider assessment, patient preference, and the potential for adverse events. Providers should consult the relevant Advisory Committee on Immunization Practices statement for detailed recommendations: http://www.cdc.gov/vaccines/pubs/acip-list.htm. Clinically significant adverse events that follow immunization should be reported to the Vaccine Adverse Event Reporting System (VAERS) at http://www.vaers.hhs.gov or by telephone, 800-822-7967.

1. **Tetanus and diphtheria toxoids and acellular pertussis vaccine (Tdap).** (Minimum age: 10 years for Boostrix and 11 years for Adacel)
   - Persons aged 11 through 18 years who have not received Tdap should receive a dose followed by Td booster doses every 10 years thereafter.
   - Persons aged 7 through 10 years who are not fully immunized against pertussis (including those never vaccinated or with unknown pertussis vaccination status) should receive a single dose of Tdap. Refer to the catch-up schedule if additional doses of tetanus and diphtheria toxoid–containing vaccine are needed.
   - Tdap can be administered regardless of the interval since the last tetanus and diphtheria toxoid–containing vaccine.
2. **Human papillomavirus vaccine (HPV).** (Minimum age: 9 years)
   - Quadrivalent HPV vaccine (HPV4) or bivalent HPV vaccine (HPV2) is recommended for the prevention of cervical precancers and cancers in females.
   - HPV4 is recommended for prevention of cervical precancers, cancers, and genital warts in females.
   - HPV4 may be administered in a 3-dose series to males aged 9 through 18 years to reduce their likelihood of genital warts.
   - Administer the second dose 1 to 2 months after the first dose and the third dose 6 months after the first dose (at least 24 weeks after the first dose).
3. **Meningococcal conjugate vaccine, quadrivalent (MCV4).** (Minimum age: 2 years)
   - Administer MCV4 at age 11 through 12 years with a booster dose at age 16 years.
   - Administer 1 dose at age 13 through 18 years if not previously vaccinated.
   - Persons who received their first dose at age 13 through 15 years should receive a booster dose at age 16 through 18 years.
   - Administer 1 dose to previously unvaccinated college freshmen living in a dormitory.
   - Administer 2 doses at least 8 weeks apart to children aged 2 through 10 years with persistent complement component deficiency and anatomic or functional asplenia, and 1 dose every 5 years thereafter.
   - Persons with HIV infection who are vaccinated with MCV4 should receive 2 doses at least 8 weeks apart.
   - Administer 1 dose of MCV4 to children aged 2 through 10 years who travel to countries with highly endemic or epidemic disease and during outbreaks caused by a vaccine serogroup.
   - Administer MCV4 to children at continued risk for meningococcal disease who were previously vaccinated with MCV4 or meningococcal polysaccharide vaccine after 3 years (if first dose administered at age 2 through 6 years) or after 5 years (if first dose administered at age 7 years or older).
4. **Influenza vaccine (seasonal).**
   - For healthy nonpregnant persons aged 7 through 18 years (i.e., those who do not have underlying medical conditions that predispose them to influenza complications), either LAIV or TIV may be used.
   - Administer 2 doses (separated by at least 4 weeks) to children aged 6 months through 8 years who are receiving seasonal influenza vaccine for the first time or who were vaccinated for the first time during the previous influenza season but only received 1 dose.
   - Children 6 months through 8 years of age who received no doses of monovalent 2009 H1N1 vaccine should receive 2 doses of 2010-2011 seasonal influenza vaccine. See MMWR 2010;59(No. RR-8):33–34.
5. **Pneumococcal vaccines.**
   - A single dose of 13-valent pneumococcal conjugate vaccine (PCV13) may be administered to children aged 6 through 18 years who have functional or anatomic asplenia, HIV infection or other immunocompromising condition, cochlear implant or CSF leak. See MMWR 2010;59(No. RR-11).
   - The dose of PCV13 should be administered at least 8 weeks after the previous dose of PCV7.
   - Administer pneumococcal polysaccharide vaccine at least 8 weeks after the last dose of PCV to children aged 2 years or older with certain underlying medical conditions, including a cochlear implant. A single revaccination should be administered after 5 years to children with functional or anatomic asplenia or an immunocompromising condition.
6. **Hepatitis A vaccine (HepA).**
   - Administer 2 doses at least 6 months apart.
   - HepA is recommended for children aged older than 23 months who live in areas where vaccination programs target older children, or who are at increased risk for infection, or for whom immunity against hepatitis A is desired.
7. **Hepatitis B vaccine (HepB).**
   - Administer the 3-dose series to those not previously vaccinated. For those with incomplete vaccination, follow the catch-up recommendations (Table).
   - A 2-dose series (separated by at least 4 months) of adult formulation Recombivax HB is licensed for children aged 11 through 15 years.
8. **Inactivated poliovirus vaccine (IPV).**
   - The final dose in the series should be administered on or after the fourth birthday and at least 6 months following the previous dose.
   - If both OPV and IPV were administered as part of a series, a total of 4 doses should be administered, regardless of the child's current age.
9. **Measles, mumps, and rubella vaccine (MMR).**
   - The minimum interval between the 2 doses of MMR is 4 weeks.
10. **Varicella vaccine.**
    - For persons aged 7 through 18 years without evidence of immunity (see MMWR 2007;56[No. RR-4]), administer 2 doses if not previously vaccinated or the second dose if only 1 dose has been administered.
    - For persons aged 7 through 12 years, the recommended minimum interval between doses is 3 months. However, if the second dose was administered at least 4 weeks after the first dose, it can be accepted as valid.
    - For persons aged 13 years and older, the minimum interval between doses is 4 weeks.

## TABLE. Catch-up immunization schedule for persons aged 4 months through 18 years who start late or who are more than 1 month behind — United States, 2011

The table below provides catch-up schedules and minimum intervals between doses for children whose vaccinations have been delayed. A vaccine series does not need to be restarted, regardless of the time that has elapsed between doses. Use the section appropriate for the child's age.

| Vaccine | Minimum Age for Dose 1 | Minimum Interval Between Doses | | | |
|---|---|---|---|---|---|
| | | Dose 1 to Dose 2 | Dose 2 to Dose 3 | Dose 3 to Dose 4 | Dose 4 to Dose 5 |
| **PERSONS AGED 4 MONTHS THROUGH 6 YEARS** | | | | | |
| Hepatitis B[1] | Birth | 4 weeks | 8 weeks (and at least 16 weeks after first dose) | | |
| Rotavirus[2] | 6 wks | 4 weeks | 4 weeks[2] | | |
| Diphtheria, Tetanus, Pertussis[3] | 6 wks | 4 weeks | 4 weeks | 6 months | 6 months[3] |
| *Haemophilus influenzae* type b[4] | 6 wks | 4 weeks if first dose administered at younger than age 12 months; 8 weeks (as final dose) if first dose administered at age 12–14 months; No further doses needed if first dose administered at age 15 months or older | 4 weeks[4] if current age is younger than 12 months; 8 weeks (as final dose)[4] if current age is 12 months or older and first dose administered at younger than age 12 months and second dose administered at younger than 15 months; No further doses needed if previous dose administered at age 15 months or older | 8 weeks (as final dose) This dose only necessary for children aged 12 months through 59 months who received 3 doses before age 12 months | |
| Pneumococcal[5] | 6 wks | 4 weeks if first dose administered at younger than age 12 months; 8 weeks (as final dose for healthy children) if first dose administered at age 12 months or older or current age 24 through 59 months; No further doses needed for healthy children if first dose administered at age 24 months or older | 4 weeks if current age is younger than 12 months; 8 weeks (as final dose for healthy children) if current age is 12 months or older; No further doses needed for healthy children if previous dose administered at age 24 months or older | 8 weeks (as final dose) This dose only necessary for children aged 12 months through 59 months who received 3 doses before age 12 months or for children at high risk who received 3 doses at any age | |
| Inactivated Poliovirus[6] | 6 wks | 4 weeks | 4 weeks | 6 months[6] | |
| Measles, Mumps, Rubella[7] | 12 mos | 4 weeks | | | |
| Varicella[8] | 12 mos | 3 months | | | |
| Hepatitis A[9] | 12 mos | 6 months | | | |
| **PERSONS AGED 7 THROUGH 18 YEARS** | | | | | |
| Tetanus, Diphtheria/ Tetanus, Diphtheria, Pertussis[10] | 7 yrs[10] | 4 weeks | 4 weeks if first dose administered at younger than age 12 months; 6 months if first dose administered at 12 months or older | 6 months if first dose administered at younger than age 12 months | |
| Human Papillomavirus[11] | 9 yrs | Routine dosing intervals are recommended (females)[11] | | | |
| Hepatitis A[9] | 12 mos | 6 months | | | |
| Hepatitis B[1] | Birth | 4 weeks | 8 weeks (and at least 16 weeks after first dose) | | |
| Inactivated Poliovirus[6] | 6 wks | 4 weeks | 4 weeks[6] | 6 months[6] | |
| Measles, Mumps, Rubella[7] | 12 mos | 4 weeks | | | |
| Varicella[8] | 12 mos | 3 months if person is younger than age 13 years; 4 weeks if person is aged 13 years or older | | | |

1. **Hepatitis B vaccine (HepB).**
   - Administer the 3-dose series to those not previously vaccinated.
   - The minimum age for the third dose of HepB is 24 weeks.
   - A 2-dose series (separated by at least 4 months) of adult formulation Recombivax HB is licensed for children aged 11 through 15 years.
2. **Rotavirus vaccine (RV).**
   - The maximum age for the first dose is 14 weeks 6 days. Vaccination should not be initiated for infants aged 15 weeks 0 days or older.
   - The maximum age for the final dose in the series is 8 months 0 days.
   - If Rotarix was administered for the first and second doses, a third dose is not indicated.
3. **Diphtheria and tetanus toxoids and acellular pertussis vaccine (DTaP).**
   - The fifth dose is not necessary if the fourth dose was administered at age 4 years or older.
4. ***Haemophilus influenzae* type b conjugate vaccine (Hib).**
   - 1 dose of Hib vaccine should be considered for unvaccinated persons aged 5 years or older who have sickle cell disease, leukemia, or HIV infection, or who have had a splenectomy.
   - If the first 2 doses were PRP-OMP (PedvaxHIB or Comvax), and administered at age 11 months or younger, the third (and final) dose should be administered at age 12 through 15 months and at least 8 weeks after the second dose.
   - If the first dose was administered at age 7 through 11 months, administer the second dose at least 4 weeks later and a final dose at age 12 through 15 months.
5. **Pneumococcal vaccine.**
   - Administer 1 dose of 13-valent pneumococcal conjugate vaccine (PCV13) to all healthy children aged 24 through 59 months with any incomplete PCV schedule (PCV7 or PCV13).
   - For children aged 24 through 71 months with underlying medical conditions, administer 1 dose of PCV13 if 3 doses of PCV were received previously or administer 2 doses of PCV13 at least 8 weeks apart if fewer than 3 doses of PCV were received previously.
   - A single dose of PCV13 is recommended for certain children with underlying medical conditions through 18 years of age. See age-specific schedules for details.
   - Administer pneumococcal polysaccharide vaccine (PPSV) to children aged 2 years or older with certain underlying medical conditions, including a cochlear implant, at least 8 weeks after the last dose of PCV. A single revaccination should be administered after 5 years to children with functional or anatomic asplenia or an immunocompromising condition. See MMWR 2010;59(No. RR-11).
6. **Inactivated poliovirus vaccine (IPV).**
   - The final dose in the series should be administered on or after the fourth birthday and at least 6 months following the previous dose.
   - A fourth dose is not necessary if the third dose was administered at age 4 years or older and at least 6 months following the previous dose.
   - In the first 6 months of life, minimum age and minimum intervals are only recommended if the person is at risk for imminent exposure to circulating poliovirus (i.e., travel to a polio-endemic region or during an outbreak).
7. **Measles, mumps, and rubella vaccine (MMR).**
   - Administer the second dose routinely at age 4 through 6 years. The minimum interval between the 2 doses of MMR is 4 weeks.
8. **Varicella vaccine.**
   - Administer the second dose routinely at age 4 through 6 years.
   - If the second dose was administered at least 4 weeks after the first dose, it can be accepted as valid.
9. **Hepatitis A vaccine (HepA).**
   - HepA is recommended for children aged older than age 23 months who live in areas where vaccination programs target older children, or who are at increased risk for infection, or for whom immunity against hepatitis A is desired.
10. **Tetanus and diphtheria toxoids (Td) and tetanus and diphtheria toxoids and acellular pertussis vaccine (Tdap).**
    - Doses of DTaP are counted as part of the Td/Tdap series.
    - Tdap should be substituted for a single dose of Td in the catch-up series for children aged 7 through 10 years or as a booster for children aged 11 through 18 years; use Td for other doses.
11. **Human papillomavirus vaccine (HPV).**
    - Administer the series to females at age 13 through 18 years if not previously vaccinated or have not completed the vaccine series.
    - Quadrivalent HPV vaccine (HPV4) may be administered in a 3-dose series to males aged 9 through 18 years to reduce their likelihood of genital warts.
    - Use recommended routine dosing intervals for series catch-up (i.e., the second and third doses should be administered at 1 to 2 and 6 months after the first dose). The minimum interval between the first and second doses is 4 weeks. The minimum interval between the second and third doses is 12 weeks, and the third dose should be administered at least 24 weeks after the first dose.

Information about reporting reactions after immunization is available online at http://www.vaers.hhs.gov or by telephone, 800-822-7967. Suspected cases of vaccine-preventable diseases should be reported to the state or local health department. Additional information, including precautions and contraindications for immunization, is available from the National Center for Immunization and Respiratory Diseases at http://www.cdc.gov/vaccines or telephone, 800-CDC-INFO (800-232-4636).
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PEDIATRICS BOARD REVIEW

PEDS

**CORE CURRICULUM**

5th EDITION

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# COMMON PEDIATRIC DISORDERS

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## Common Pediatric Disorders

# COMMON CONCERNS OF PARENTS

## COLIC

Colic is a syndrome of findings believed to be a specific condition, but this condition has yet to be fully understood. Clues to colic include the following:

- Crying in the later afternoon/evening that peaks in the 2nd month and wanes in the 3rd–4th month.
- Physical manifestations—legs over abdomen, clenched fists, pained facies, gas, spitting up, and lack of response to soothing. But, in general, the physical exam is normal!
- Crying bouts that are prolonged and paroxysmal—begin and end without warning and are unrelated to the environment.

Classically, 4 characteristics may appear in real life and on the Boards:

1) Paroxysmal crying
2) Qualitatively different crying from the baby's normal crying
3) Hypertonic positioning
4) Inconsolable

A simple way to suspect colic is through Wessel's rule of 3s: Crying > 3 hours/day for > 3 days a week for > 3 weeks. It is estimated that colic occurs in 8–50% of infants.

The difficulty is determining if the crying is due to colic or an organic cause. Less than 5% of infants undergoing evaluation for excessive crying have an organic etiology—but, you don't want to miss these 5%! For infants < 1 month, you must rule out other causes of irritability (e.g., sepsis, intracranial bleed). If the child is > 4 months, it is not colic. Other criteria that point more toward organic causes include the following:

- A high-pitched cry that is not diurnal.
- A big hint for the Boards—An infant described as a poor feeder who cries excessively and becomes diaphoretic during feeds is likely to have an anomalous left coronary artery.
- On physical exam and history, there are findings besides crying; e.g., corneal abrasion, otitis media, mouth ulcers, hair tourniquets, skeletal infection or fracture. 2 favorite nonintentional trauma "look-alikes" that present with crying (that may be misinterpreted as colic) are due to bone pathology—pseudoparalysis of a limb associated with congenital syphilis and Caffey disease (infantile cortical hyperostosis).
- Later onset (starts in the 3rd month) and follows switching from breast to formula feeds—indicating that cow's milk protein intolerance is likely.

Treatment for colic is difficult. Most cases resolve by age 3 months, but about 1/3 will persist to 4 months and then resolve. If it does not resolve after 4 months, search for organic causes. Provide parents with information about the typical pattern and the characteristic lack of response to soothing. There is limited data that whey hydrolysate milk may be helpful. There is good evidence that giving lactase or simethicone is not helpful. Reassure them of both the typical excellent outcome and the fact that colic is not due to being "bad parents." Environmental factors, such as music, singing, "white noise," or car rides, help some infants. Caution parents against the idea of using washer/dryer vibrations to alleviate colic symptoms because of the risk that the child could fall off the appliance (or inadvertently be left in the dryer—just a joke to see if you are still awake). Also, caution parents about shake injuries/shaken baby syndrome. It is essential that caretakers have someone to relieve them of dealing with the colicky infant on a periodic basis.

## SLEEPING PROBLEMS

### Normal Sleep Development

By age 3 months, 50–70% of children will sleep from midnight to 5 a.m. without parental intervention. This percentage increases to 80% by age 5–7 months, and to 85–90% by 1 year. Between ages 6 and 12 months, about 50% of those who have been sleeping during the night will have periods of wakefulness and crying—thought to be related to "separation anxiety" (a new cognition) and object permanence. Persistent, disruptive sleeping occurs when children are not allowed or not able to handle the normal arousals (end of sleep cycle, REM sleep, and non-REM sleep). Most sleep disturbances are due to intervening parents who are concerned inappropriately about normal arousals! See Table 2-1.

### Sleep Associations

Sleep associations are behaviors/expectations the child develops based on relationships created in her world, most often by parents. A common sleep association is when a parent holds, rocks, or sings to the child at bedtime. The child associates falling asleep with this behavior. When the behavior does not occur, the child has difficulty falling asleep. It is particularly a problem if the child wakes up during the night—she cannot soothe herself to sleep, because she is accustomed to the stimulation/reinforcement provided by her parents.

Feeding during the night after 6 months of age is another learned association. It is not physiologically dictated, but infants learn it through reinforced behavior.

Other sleep associations—more correctly, child-waking associations—occur when parents take the baby from the crib, watch television, play with the child, or do other activities in the child's room as a response to the child waking during the night.

Psychosocial factors can aggravate poor sleep patterns. Family stressors, such as changes in jobs, moves, marital discord, travel, etc., can manifest as sleep

**Table 2-1: Normal Sleep Durations and REM Patterns**

**Infants:**

- Longer sleep duration (16–18 hours per 24 hours)
- REM sleep occurring at sleep onset
- Increased proportion of REM sleep
- REM-NREM cycle much shorter in duration as compared with older individuals

**Children:**

- Onset via NREM sleep
- NREM sleep occupying approximately 75% of total sleep time
- REM and NREM sleep alternating throughout the night with a period of 90 to 100 minutes, and a progressive lengthening of the duration of REM sleep periods in the final one-third of the night

**Sleep in adolescents is further characterized by:**

- Sleep requirement of about nine hours
- Decrease in slow-wave sleep beginning in puberty and continuing into adulthood
- Physiological shift in sleep onset to a later time

disturbances—likely due to the parents' preoccupation with the life stressors instead of carrying through the usual child management strategies.

Co-sleeping (sleeping in a parental bed) occurs with 35–55% of children < 4 years of age. Cultural and ethnic differences are common; the prevalence is much higher in African-American and Latino families. However, most professional pediatric organizations discourage this practice due to the increased risk of SIDS, although in July 2010 a new study showed data that conflicted with this hypothesis.

## Nightmares

Nightmares are disturbing dreams that occur during REM sleep. They are usually followed by awakening and distress about the dream. Nightmares are common; peak age of onset is between years 3 and 5. Nightmares tend to occur after several hours of sleep and do not usually cause a child to get out of bed. Parental reassurance is the best response. Recurrent nightmares with frequent night waking usually indicate significant distress in a child's daytime life. (Note: Nightmares concurrent with studying for the Boards are also common, especially those "showing up for the test naked" dreams.)

Children with nightmares need to be reassured that the dream is over and that they are safe in their bed. Also, talking about the dream the next day may be helpful. Frequent nightmares may be a reaction to a serious stressor occurring in the household; you should take care to consider if this is the case, and what you can do to lessen the stressor.

## Night Terrors

Night terrors differ from nightmares. Night terrors produce a distinct non-REM parasomnia—an abrupt arousal from stage 4 non-REM sleep to near arousal. Most occur during the first third of the night. The child appears to be awake but is unresponsive and unaware of the parent's presence, difficult to arouse, cries intensely, is often diaphoretic, and appears disoriented. Later, the child doesn't remember the episode (hmm … reminds me of sitting in anatomy class). Night terrors are more common in males and occur in ~ 1–6% of children. Night terrors peak at 5–7 years of age, but may occur as early as 18 months. Again, parental reassurance is the best therapy. Night terrors can persist into adolescence. Frequently, you will find a family history of night terrors in your evaluation.

Parents should just observe night terrors without awakening the child. In fact, awakening a child with a night terror will prolong the attack. Some have noted that recurrent night terrors occur at about the same time of the night, and a "preemptive awakening" before the usual time of the night terror can actually break the cycle and prevent further terrors from occurring. They can be triggered by stress, sleep deprivation, and seizure/ADHD medications.

## Sleepwalking (Somnambulism)

Sleepwalking is another phenomenon where the child appears to be awake but isn't. It also occurs during stage 4 non-REM sleep. It tends to occur in children between ages 4 and 8 years, with a prevalence of about 15%.

For sleepwalkers, encourage parents to ensure the environment is safe, to observe the child, and to gently lead the child back to bed. The same process of "preemptive awakening" prior to the usual sleepwalking event can be successful in stopping the behavior.

## Sleeptalking (Somniloquy)

Sleeptalking can occur during any stage of sleep, from partial arousal to REM sleep. It is common and can persist throughout adulthood.

Sleeptalkers require no specific intervention (although my wife seems to prefer a strong kick to my back).

## Treatment and Management of Sleep Problems

As parents and practitioners, we vary on treatment approaches, but what you need to know is what the books say to do, because that is what the ABP expects you to know and do (even though your "real world" may be different).

**QuickQuiz**

- What is the upper age limit after which colic should not be considered as a diagnosis?
- Do nightmares occur during REM or non-REM sleep? What about night terrors?

First, a regular daily routine is very important. Make sure children are getting enough sleep because sleep disorders are more common in children who do not get enough restful sleep.

Infants should be in a crib that is not located in the parents' room. Babies should be put down when they are drowsy and not yet asleep. Middle-of-the-night feedings should stop by age 4–6 months.

Parents can best handle night waking in older infants by ignoring it, but they will need reassurance that ignoring is okay. It often resolves after a week, with the infant falling back to sleep without intervention. Occasional verbal reassurance can be useful, but encourage parents to not pick up and hold the infant unless there are unusual circumstances.

Older children (toddlers to preschoolers) should have a regular pre-sleep routine—bathing, changing clothes, brushing teeth, reading books, etc. Night-lights are helpful for those afraid of the dark. Meet attempts by the child at bedtime to "get this" or "do that" with a gentle but firm "No, it is bedtime." Younger, school-aged kids generally enjoy this time to get special attention from the parent—to hear a story, to read together, or to talk about the activities for the next day.

Adolescents generally want to be left alone and arrange their own bedtime activities. However, parents need to monitor and (attempt to) set limits if this is interfering with daily living (school, job, sports, etc.). Usually the non-sleep activities include television, video games, computer, or phone (texting) time and should be "off-limits" at bedtime to facilitate a calming nighttime routine.

### Obstructive Sleep Apnea (OSA)

OSA is characterized by repeated episodes of prolonged upper airway obstruction during sleep despite continued or increased respiratory effort, resulting in complete or partial cessation of airflow at the nose and/or mouth, as well as disrupted sleep. Intermittent hypoxia and frequent sleep arousals are common and result in long-term sequelae. Nasal (choanal stenosis/atresia, deviated septum, rhinitis, polyps, etc.), oropharyngeal (adenotonsillar enlargement, macroglossia, cleft palate repair, masses), and craniofacial (micrognathia, trisomy 21, Pierre Robin sequence, achondroplasia, etc.) anatomic abnormalities can predispose to OSA.

Increased weight correlates greatly with increased incidence of OSA. The prevalence of OSA documented by overnight sleep studies is 1–4%.

Nocturnal symptoms may include the following:

- Loud, frequent, and disruptive snoring
- Breathing pauses
- Choking or gasping arousals
- Restless sleep
- Nocturnal diaphoresis

Daytime symptoms may include the following:

- Mouth breathing
- Dry mouth
- Chronic nasal congestion or rhinorrhea
- Hyponasal speech
- Morning headaches
- Difficulty swallowing
- Poor appetite
- Daytime sleepiness and drowsiness (but much less common in children than adults)
- Difficulty with morning waking

Mood changes may include:

- Increased irritability
- Mood instability
- Low frustration level
- Depression
- Anxiety
- Overlap with ADHD diagnostic criteria

The gold standard for diagnosis is an overnight polysomnogram. Treatment is not standardized in children. A majority of cases of OSA will start therapy with adenotonsillectomy if there is evidence of increased glandular size. Other therapy after this includes weight loss and/or positional therapy. Continuous or bilevel positive airway pressure (CPAP or BiPAP) is the most common therapy in adults and is also successful in children.

## LANGUAGE DEVELOPMENT

Language development is obviously very important—and the ABP thinks so too—so plan to memorize and/or understand when appropriate language ability is supposed to occur. Definitely plan on a few questions about this on the Board exam.

Table 2-2 lists milestones of "expressive" language development.

On the exam, the ABP will ask you what is "normal" and what is "abnormal." It is a pain to remember these. If you are a recent new parent, you may be able to remember these developmental things first-hand. If not, up to the time you take your exam, pay special attention to the developmental history when you see children as patients. Get it straight in your mind what is normal for

a 2-year-old, a 3-year-old, etc. It will help you tremendously on the ABP exam! Table 2-3 lists observations to be concerned about for speech and language development as these relate to specific age groups.

From the table, know that a 2-year-old should be using 2-syllable words, and that a 4-year-old should be able to tell a simple story. These standards are easy for you to use in practice, too.

"Late talking" generally defines any set of circumstances in which the verbal abilities of a child are noticeably behind the norm. What are its probable causes? A common cause is hearing impairment—which dictates a formal assessment by an audiologist. This is not something to be done in a pediatrician's office, where the conditions are less than ideal. Another common cause is developmental delay, which could be due to a variety of etiologies. Other causes of "late talking" include mental retardation, specific language disorders, autism, dysarthria, dyspraxia, maturation delay, and neglect. Remember: For the Boards, when presented with a child with a speech problem, always check the hearing first! Rationalizations such as a bilingual home, gender, family history of speech delay, and the child does not speak but understands everything, should not be used to "explain away" delayed speech.

Table 2-2: Expressive Language Development

| | |
|---|---|
| Birth to 12 months | Coos, laughs, squeals, babbles, turns to orient to name, different cries noted, Mama, Dada (nonspecifically and specifically), polysyllabic babbling, gives toy with gesture points, shakes head, says as least 1 word clearly, can identify objects, points, shakes head |
| 12–18 months | Says 2 words other than Mama or Dada, gives toy on request without gesture, combines jargons and gestures, uses 15–20 words, 2–3 word phrases, follows simple instructions, can identify 4 body parts, uses gestures well, speaks in a way that immediate household family members can understand |
| 18–30 months | Expanding vocabulary, 100–200 words, 2–3 word phrases, more fluency—less stuttering, uses personal pronouns: me/mine, identifies 6 body parts, speaks in present tense, speech about 25% intelligible to strangers, understands prepositions: on/off/in/out/under/behind |
| 3 years | Speaks in 3–4 word sentences, talks in short paragraphs, speech is 75% intelligible to strangers, uses plurals and pronouns, uses "what" and "who" questions, can identify 2 colors |
| 4 years | Speaks in 4–5 word sentences, speech 100% intelligible, talks in full paragraphs, able to tell a story or explain a recent event, uses past tense, sings nursery rhymes, says first and last name, identifies gender, knows 5–6 colors, asks "why" questions |
| 5 years | Able to define simple vocabulary words, understands and able to answer knowledge questions |
| 6 years | Transitions from preoperational to operational thinking, reads by word recognition, can repeat a complex sentence |

## HEARING PROBLEMS

Deafness is defined as hearing loss > 90 dB. This results in the inability to distinguish between elements of spoken language. "Mild" hearing loss = 25 dB loss, but even a 15 dB loss can result in problems with speech perception, especially during early childhood.

Conductive hearing loss (more common) is due to disruption of mechanical components required for the transduction of sound wave energy. In addition to cerumen impaction, the other most common cause of conductive hearing loss is fluid in the middle ear—most commonly secondary to an otitis media. Children with this type of loss can hear speech, but fluid-caused distortions can lead to problems in early language discrimination.

Table 2-3: Clues to Abnormal Speech and Language Development by Age

| | |
|---|---|
| 9 months | No babbling |
| 15 months | No first words |
| 18 months | No consistent words |
| 18–24 months | Child uses only a few words, hardly any phrases |
| 2 years | Child cannot follow simple directions<br>Child points instead of speaks<br>Child is not using 2-syllable words, or combining words<br>Speech is difficult for parents to understand |
| 2½ years | Child cannot be understood most of the time<br>Child frequently omits first or last consonant of a word<br>Child cannot understand 2-step directions<br>Child cannot pronounce: b, h, m, n, p, w |
| 3 years | Child cannot repeat a 4- or 5-word sentence<br>Speech is difficult for strangers to understand |
| 3½ years | Child cannot name specific objects easily<br>Child omits words in sentences<br>Child cannot pronounce d, f, g, k, t |
| 4 years | Child cannot tell a simple story |
| 5 years | Child cannot pronounce l, j, v, ch, sh |
| 6 years | Child cannot pronounce r, s, z, st, th |
| 5–6 years | Poor memory skills: inability to learn colors, numbers, shapes, alphabet |

**QuickQuiz**

- Know Table 2-2!
- Would you be concerned if a 2-year-old pointed at a ball without also saying the word "ball" in order to receive the ball? Why or why not?
- True or false? You should be concerned if a 5-year-old cannot pronounce the letter "d" correctly in words.
- What is the most common cause of conductive hearing loss in children?
- What is the most likely etiology for severe or profound hearing loss?

About 65% of children have some degree of intermittent conductive hearing loss. This hearing loss is limited to sounds at 50 dB or lower, because sounds louder than this can be conducted directly by bone to the cochlea.

Sensorineural hearing loss (less common) is a problem due to dysfunction of the sensory epithelium, the cochlea, or the neural pathways to the auditory cortex, using the 8th cranial nerve and other connections. Severe or profound hearing loss is always sensorineural and most often affects high frequencies. Severe or profound hearing loss affects about 1–2/1,000 children at birth; an additional 2–3/1,000 experience an acquired, severe hearing loss in childhood.

Cortical hearing problems can also occur. These result in the inability, or impaired ability, to perceive or process sounds.

Deafness can be due to either an isolated event or a syndrome, such as Treacher-Collins, Alport, Crouzon, Waardenburg, Usher, or Down syndrome. One form of the prolonged Q-T syndrome (Jervell and Lange-Nielsen syndrome) is associated with sensorineural hearing loss—remember this if a Board question describes a patient with syncope and a history of hearing loss. Deafness is inherited in ~ 50% of cases. 80% are inherited as autosomal recessive, 18% autosomal dominant, and 2% are X-linked recessive.

Infectious etiologies are the other significant factor in deafness. Today, CMV (most common and causes hearing loss in 60% of symptomatic infants and 7% of asymptomatic) and toxoplasmosis account for the majority of infectious causes—in contrast to decades ago, when rubella was one of the most common causes. (Rubella as a cause is now rare because of an effective preventive vaccine.) Bacterial meningitis is still a common cause of deafness and occurs in about 10% of those affected. Immunizations (both *Haemophilus* and pneumococcus) have reduced this in recent years.

Prolonged exposure to loud noise is a common cause of high-pitched hearing loss in adolescents.

Table 2-4 lists factors identified by the Joint Committee on Infant Hearing as risk factors for neonatal hearing loss.

Around 50% of infants with sensorineural hearing loss will have one of the risk factors listed in Table 2-4. However, since this also leaves 50% of infants not exhibiting these risk factors, many states have initiated universal screening for infants.

Screening is done with Evoked Otoacoustic Emissions (EOAE) most commonly done first, then with automated Auditory Brainstem Response (ABR) systems if the baby fails EOAE testing.

Other children who should be screened are those with a persistent, middle ear effusion that has lasted > 2 months. Any loss of hearing for a prolonged period of time during the critical period of speech acquisition can lead to a speech problem.

The key to success in treating hearing loss is early diagnosis and intervention.

## Tympanograms

We will present several common tympanograms. Know these!

Type A, normal tympanogram (Figure 2-1):

- Peak +50 to –150 mm
- Peak compliance (*y* axis) between 0.2 and 1.8 cc
- Absence of middle ear pathology
  - Intact and mobile TM
  - Normal eustachian tube
  - If hearing loss, likely sensory-neural

**Table 2-4: Factors Associated with Hearing Loss in Neonates**

| |
|---|
| Family history of sensorineural hearing loss |
| Congenital infection |
| Presence of craniofacial anomalies |
| Birth weight under 1,500 grams |
| Neonatal jaundice resulting in exchange transfusion |
| Ototoxic medications (furosemide, aminoglycosides) |
| Bacterial meningitis |
| Apgar scores of 3 or less at 5 minutes |
| Physical findings consistent with a syndrome associated with hearing loss |

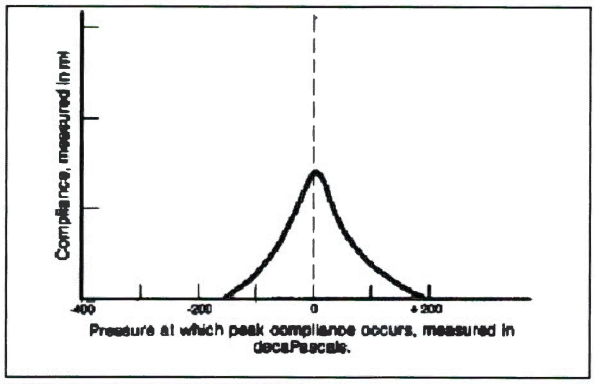


**Figure 2-1:** Type A, Normal Tympanogram

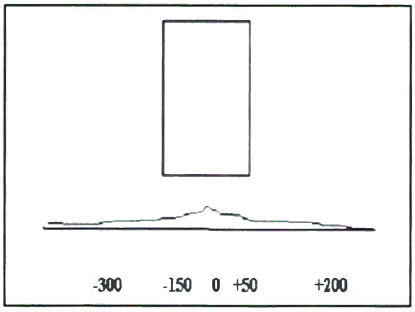


**Figure 2-2:** Type A(s), Shallow

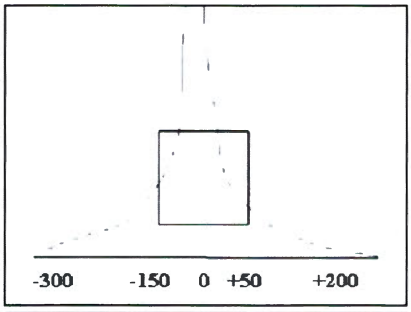


**Figure 2-3:** Type A(d), Disarticulation

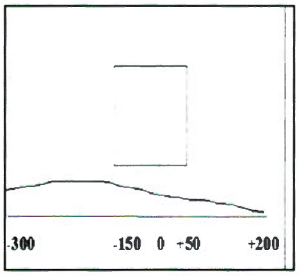


**Figure 2-4:** Type B, Retracted, Poorly Mobile

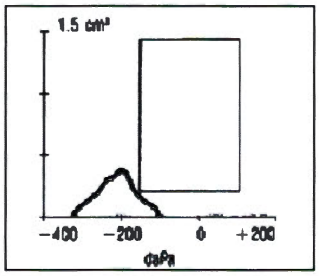


**Figure 2-5:** Type C, Negative Pressure

Type A(s), shallow (Figure 2-2):

- Peak pressure curve normal in position
- Peak compliance very low (below 0.2 cc)
- Associated with ossicular fixation or TM scarring
- Not due to middle ear effusion
- May result in hearing loss
- Can be caused by otosclerosis

Type A(d), disarticulation (Figure 2-3):

- Peak pressure between +50 and –150 cc (normal)
- Peak compliance very high
- Associated with ossicular disarticulation
- May result in hearing loss

Type B, retracted, poorly mobile (Figure 2-4):

- Peak is absent or poorly defined
- Negative middle ear pressure (peak pressure is shifted left)
- Maximum compliance below normal
- Suggestive of fluid behind middle ear

Type C, negative pressure (Figure 2-5): [Note: This is the one most likely to appear on the Boards!]

- Clear peak
- Negative middle ear pressure (left shift: peak pressure negative)
- Peak compliance may be normal
- Diagnosis: eustachian tube dysfunction
- Conductive loss

## VISION PROBLEMS

### Overview

The most prevalent vision disorders are: amblyopia, strabismus, significant refractive error, color-vision defects, and ocular disease. In the U.S., the leading causes of blindness in children are cortical visual impairment, retinopathy of prematurity, and optic nerve hypoplasia. The leading cause of "acquired" blindness is ocular trauma. The #1 cause of such trauma is accidental or intentional injury by another child. The next most common cause is sports-related injuries.

### Normal Visual Development

In formal terminology, here are some important basics:

1) Visual fixation occurs in the newborn and achieves accuracy by 6–9 weeks.
2) By 3 months of age, infants have the ability to follow an object.
3) Optokinetic nystagmus and vestibular ocular reflex are normal involuntary eye movements that provide stability of visual images.

**QuickQuiz**

- Know the common tympanograms.
- Are most term infants near- or far-sighted at birth? What about premature infants?
- Define strabismus.
- What is the most common cause of amblyopia?
- How common are color-vision abnormalities in boys compared to girls?
- What is the most common etiology of retinopathy of prematurity?
- What is the most common malignant ocular tumor in childhood?

4) Accommodation is the ability to focus the intraocular lens for near-viewing and is present at birth; however, it is inaccurate until age 2–3 months.
5) Most term infants are hyperopic (farsighted) at birth, while premature infants are myopic (nearsighted). The fovea reaches maturity at 4 years of age, and children reach 20/20 vision by 5 years. (Newborns start with 20/400—hmm, how do you get a newborn to do the wall chart?)
6) Color discrimination occurs by 2 weeks of age and improves over the next 3 months.
7) Fine-depth perception occurs at ~ 3 months and is near adult functionality by 6 months.
8) An abnormal red reflex may indicate a number of abnormalities (cataracts, glaucoma, retinoblastoma, strabismus) and should be referred immediately.

## Strabismus

Strabismus is the misalignment of the eyes (visual axes), in which one and/or the other eye is turned in (esotropia), out (exotropia), up (hypertropia), or down (hypotropia). The misalignment can occur continuously or intermittently. The most common forms in childhood are the infantile and accommodative esotropia. Amblyopia commonly occurs with esotropia. Children with prominent epicanthal folds and/or a wide-bridged nose—both of which may "hide" the nasal sclera—often appear to have strabismus (pseudostrabismus) but, on formal testing, are noted to have a symmetric light reflex. (See cover/uncover test in Growth and Development.)

## Amblyopia

Amblyopia occurs at a rate of 75,000 new cases each year in 3-year-olds and is present in 1–4%. Amblyopia causes more vision loss than any other disease in individuals < 45 years. Amblyopia is loss of visual acuity that is not due to ocular pathology and is not correctable with glasses or contact lenses. It is caused by disuse or misuse of visual pathways during the critical period of visual development. Amblyopia may result from early childhood refractive disorders, strabismus, cataracts, or corneal opacities. The most common causes of amblyopia are strabismus and anisometropia, an unequal refractive error between the eyes.

## Color-vision Defects

The ability to match colors is present by age 2 years (hmm … some of us never get that, I guess). Abnormal color vision occurs in ~ 8–10% of boys and < 0.5% of girls and is due to X-linked inheritance protan and deutan deficits (blue-green). Other rare defects that affect color vision include retinal or optic nerve disorders.

## Cortical Visual Impairment

Cortical visual impairment is vision loss due to damage to the geniculostriate pathway, which is composed of the visual cortex and optic radiations. It presents as reduced vision, absence of optokinetic nystagmus, and intact pupillary light responses. Its causes are numerous, including hypoxia, meningitis, encephalitis, metabolic disease, head trauma, and hydrocephalus. The most common cause is hypoxia. Usually, these children have other, associated abnormalities, including cerebral palsy, seizures, or paralysis.

## Retinopathy of Prematurity (ROP)

Retinopathy of prematurity is a disorder of immature retinal vasculature and is provoked by excess oxygen use in premature infants. Today, severe ROP occurs mainly in infants born at < 30-weeks gestation and with birth weights < 1,250 grams. Attempts to prevent ROP have been largely unsuccessful. Current AAP policy is to screen infants < 1,500 g or < 32-weeks gestation.

## Optic Nerve Hypoplasia

Optic nerve hypoplasia is a nonspecific finding due to damage of the visual system before it is fully developed. It is associated with maternal diabetes, alcohol abuse, and maternal exposure to toxins. You might also see optic nerve hypoplasia with hypothyroidism, growth hormone deficiency, and neonatal hypoglycemia. Optic nerve hypoplasia is an example of a midline facial defect—other such defects include a single central incisor and cleft lip/palate. Also, speaking of midline defects, if a Board question describes a male with a microphallus or undescended testicles, remember that this may be associated with hypopituitarism and/or growth hormone deficiency.

## Retinoblastoma

Retinoblastoma is the leading malignant ocular tumor of childhood. It occurs at a rate of 1/16,000. ~ 50% of all cases are familial, but if bilateral, then 100% are familial. Retinoblastoma is associated with chromosome

13q- (deletion); affected children also have mental retardation, skeletal anomalies, and increased risk of additional tumors most commonly osteogenic sarcoma. The tumor comes from the primitive retinal cells, and you will see them most in children < age 4 years. With treatment, survival is > 90%. The most common symptom/sign is a white pupil, known as leukokoria. Strabismus, glaucoma, and poor vision can also occur, but are less common presentations.

## BIRTH OF A SIBLING—RESPONSES

Birth of a sibling almost always evokes some feelings of hostility by the older sibling toward the new baby. Some children will exhibit signs of developmental regression, such as thumb sucking or regressive toilet training. Others may become sad or withdrawn, and still others may develop aggressive behaviors at home or at day care.

Some parents ask, "When is the best time to have another child?" Experts disagree, but a 2-year interval appears to be optimal in some anecdotal data. Note: There is no definitive data on this question at this point.

It is important to encourage parents to be open with their older child(ren) about the pregnancy and to prepare them for its impacts. Allow the older child to participate in the planning before the delivery and to be involved in taking care of the newborn when he or she arrives at home. A small gift from the "new baby" is a nice touch. If changes in bedrooms or beds are necessary because of the birth, carry these out well in advance of the delivery. Be sure to not associate the changes with the arrival of the new baby.

If he is present, encourage the father to increase his attention and involvement with the older child. Studies show that this involvement provides the best intervention to ensure comfort and reassurance for the older child. If no father is present, suggest that another relative or other adult close to the family assume the role of providing the older child with extra attention and encouragement.

## TEMPER TANTRUMS

Temper tantrums occur in nearly all 2- to 4-year-olds. Occurrences may be daily in ~ 20% of 2-year-olds and 10% of 4-year-olds. These can manifest in many ways. Breath holding is common in infants and children and usually is associated with a temper tantrum caused by an environmental event. It appears to be autosomal dominant with reduced penetrance! Therefore, family history is frequently positive for a history of breath holding or fainting. Temper tantrums usually begin in the 2nd year, but may appear as early as 7 months! They usually resolve by 5 years of age.

There are 2 types of breath-holding temper tantrums:

1) A cyanotic form, in which the face turns blue until breathing returns
2) A pale form, in which the face turns pale due to vasovagal syncope

Cyanotic breath-holding spells are usually preceded by several shrill "cries" followed by a period of prolonged expiratory apnea. The child rapidly loses consciousness and becomes cyanotic. They often occur when the child is upset by an event—e.g., he is told "no" to a request for another cookie or is asked to put away a toy. Pale, or pallid, forms have a rapid onset, are not usually associated with a cry, and follow a painful event—like if a child ran into a tree while flying a kite! The child stops breathing, rapidly loses consciousness and becomes pale and hypotonic. Syncope may occur, especially with the pale form, at the onset of crying. The period of not breathing is brief, and the child will then have spontaneous respiration. With the syncope, tonic-clonic movements can occasionally occur.

Breath-holding spells are benign—there are no long-term effects or brain injury. But they scare the heck out of the parents! Aim treatment at preventing situations that may induce an attack; reassure parents that these spells are not life-threatening or dangerous.

## DISCIPLINE

Effective discipline is a difficult and common subject of query to pediatricians. Discipline is very important for normal development and is central to the emotional stability of a child. Modeling positive behaviors is the most effective means of discipline. Parents do this with positive language and actions. Punishments should be consistent from one family member to another (this is especially important—and sometimes difficult—among extended families living in the same household), and from one similar episode of misbehavior to another. Discipline should occur as quickly as possible after the event that triggers it.

For the ABP exam, any type of physical punishment, such as spanking, is unacceptable. No matter how common and acceptable corporal punishment is in certain cultures and among certain ethnic groups, for the Board exam, spanking is an unacceptable punishment! "Time-outs" are a favorite, and many consider loss of privileges (no television, no sleep-overs, etc.) as sending a strong message to older children—so go with these types of options on the Boards!

## SEPARATION ANXIETY

Separation anxiety—often exhibited as "clingy" behavior toward the mother—usually becomes noticeable at about age 6 months. It is especially prominent at about 9–18 months and begins to lessen by 3 years of age. These children are afraid to leave their mothers, are

**QuickQuiz**

- How does retinoblastoma usually present?
- When a new sibling is about to arrive, what should the father do?
- What are the brain injury risks of breath-holding spells?
- Does the ABP examination encourage spanking as a form of punishment?
- At what age does separation anxiety become noticeable? When does it begin to lessen?
- What complaints are commonly associated with school refusal?
- At what age(s) does body rocking occur?
- At what age(s) does head banging occur?
- True or false? In most children with head banging, radiologic and neurologic studies are required to evaluate the condition.

afraid to be with strangers, and/or are fearful of new situations. Many children > 3 years of age will continue to have uneasiness about unknown or new situations, but most children can handle the feelings internally or verbally without crying or other symptoms. Parents should be encouraged to say goodbye quickly when leaving their children and not prolong the event or promptly return (sometimes multiple times!) when and if their child cries.

Attachment styles (developed by Mary Ainsworth): A majority of children are considered "secure"—they will protest the mother leaving but can be consoled and distracted with toys, etc., in the room, and then they will seek contact with the mother on her return to the room. ~ 20% of children are termed "avoidant"—they don't notice the mother coming or going, and, when she returns, they move away from the mother and continue to play. About 10% are "ambivalent"—these kids suffer great distress when the mother leaves the room, cannot be consoled while the mother is gone, and cannot easily be consoled on the mother's return. The final 10% are considered "disorganized"—they exhibit both the avoidant and ambivalent behaviors.

## SCHOOL REFUSAL

School refusal is not to be confused or equated with truancy. In truancy, the child is not at home or at school, and the parent is unaware. With school refusal, the parent is aware because the child is home. School refusal usually occurs in association with somatic complaints—headache, stomachache, etc. School refusal is defined as missing school for 2–3 days/week for at least 2 weeks. You may find anxiety, phobias, and behavior disorders (depression, oppositional behavior) in these children. Family disruption (divorce, domestic violence, illness, or death) is often associated with school refusal.

School refusal occurs in about 3–5/1,000 children overall, with an increased prevalence in high school of 1–5%.

School refusal is most common in the early teen years and more common in boys than girls. There is no difference between races or socioeconomic statuses.

Encourage parents to set strong expectations that children attend school. Although a parent should never dismiss physical complaints, the focus should be on getting the child to school in spite of the physical complaints. Parents can enroll the help of teachers, the school nurse, or the school social worker (if available) to facilitate school attendance while reducing the child's anxiety. Setting a clear expectation that the child will go to school must be part of the plan to address school refusal. Also, you need to address anxiety and other underlying issues if they are the reason for school refusal.

## "BAD" HABITS AND UNDESIRABLE BEHAVIORS

### Body Rocking

Body rocking occurs at ~ 6 months of age in 5–20% of children. The rocking occurs in either the sitting or crawling position, and can be quite vigorous. Some kids have been known to rock their cribs across the room or damage walls/cribs with the banging. It is most common around bedtime and lasts ~ 1/2 hour. Body rocking usually stops by age 2–3 years—it rarely continues into adolescence.

### Head Banging

Head banging occurs in 5–15% of "normal" children. The onset of head banging is usually ~ 8–9 months of age, and the behavior stops by age 4 (unless they join a rock-and-roll band in high school). It occurs much more frequently in males and most commonly at bedtime or in the middle of the night. The episodes can be very short, or they may persist for 3–4 hours!

Multiple studies have been done on these kids, but no EEG or other neurologic abnormalities have been noted. Reassure parents that this is normal behavior and that no permanent damage will occur. Padding areas of favorite banging is useful to minimize bruising or callus formation. Children with mental retardation or autism who head bang may require helmets or medications to lessen the head-banging activity and prevent injury. Remember: Restricted, repetitive, or stereotypic patterns of behavior may be an indication of an autistic-spectrum (pervasive developmental delay) disorder.

### Thumb Sucking

Thumb sucking has been described *in utero* as early as 28-weeks gestation and can continue until age 5 years or so. Peak thumb sucking age is 18–21 months; most stop by age 4. Thumb sucking occurs in anywhere from 10–35% of children. Thumb sucking that persists into adolescence is more common in girls than boys and may indicate underlying psychological problems. Prevalence of thumb sucking in breastfed infants is higher than in bottle-fed infants, but the duration of thumb sucking is lower in the breastfed group.

Thumb sucking beyond age 4 years merits referral for dental evaluation because of the significant potential for dental problems. Before the child turns 4, reassure parents that thumb sucking is natural and usually resolves spontaneously. Attempts by parents to stop the behavior may actually reinforce it. Positive feedback when the child is not sucking their thumb is helpful. Some recommend hypnotherapy or adversive therapy, such as applying bitter nail polish to discourage sucking, which has also been successful. Older children may require a dental appliance to prevent any comfortable insertion of the thumb.

### Nail Biting (Onychophagia)

Nail biting (fancy name: onychophagia) is a common habit of children and adults. It includes biting of the nail itself, the cuticles, and/or soft tissue, and frequently leads to irritation, bleeding, and infection. Nail biting is most common between ages 10 and 18 years. About 50% of children have the habit at some time in their childhood. Between ages 5 and 10 years, boys and girls are equally affected, but after age 10, boys are more commonly affected. About 10–20% of men > 25 persist in biting their nails.

Again, aim treatment at positive reinforcement. Encourage parents to praise children during the periods in which they don't bite their nails.

### Clinching / Grinding of Teeth (Bruxism)

Grinding or clinching of the teeth produces a high-pitched, annoying sound; this is typically nocturnal, during REM sleep. Prolonged grinding can cause tenderness in the muscles of the mouth, temporomandibular joint pain, tooth damage, tension headaches, face pain, and neck stiffness in adolescents. In children, bruxism is usually self-limited and does not require intervention. Bruxism is most common in boys and appears to be familial. Teeth and supporting structures can be damaged over time. For adolescents, recommend the use of a splint or bite guards, which are created by a dentist.

### Hair Pulling (Trichotillomania)

Less common than the other habits we have discussed so far, trichotillomania is the inability to resist pulling out one's own hair. The child usually removes hair from the scalp and creates bald spots, but some pull eyelashes, eyebrows, and pubic hair. Some children will chew the hair and then swallow it—called trichophagy—and over time large amounts can result in a hair bezoar, known as trichobezoar. Most believe this behavior to be within the obsessive-compulsive disorders.

Most affected are girls. There are 2 categories within this disorder:

1) Early onset: Presents < 5 years of age and then with a long history of hair pulling. These children pull hair when bored or tense.
2) Late onset: Presents in later childhood, adolescence, or adulthood. Other pathologies, such as depression, anxiety, history of physical and/or sexual abuse, bipolar disorder, substance abuse, and personality disorders are commonly associated with the late-onset group.

Alopecia is common and must be differentiated from alopecia areata (which is usually of sudden onset, well circumscribed, and has non-scarring areas of hair loss versus irregular patches with hairs of various lengths in trichotillomania), tinea capitis, syphilis, telogen effluvium, and traumatic/traction alopecia. Treat early-onset trichotillomania with positive-reinforcement techniques and use time-outs for episodes. The late-onset disorder is difficult to treat and usually involves dealing with the underlying psychiatric disorder. Note: Iron deficiency anemia has been associated with trichotillomania and trichophagia, so consider screening for iron deficiency, which can cause pica. Also consider medications that cause hair loss (e.g., anticonvulsants and isotretinoin).

## TIC DISORDERS

### Tics

Tics are involuntary, purposeless stereotyped movements, gestures, or utterances that are usually briefly suppressible. The areas most commonly affected are the muscle groups of the eyes, mouth, face, and neck. Variants of these disorders include copropraxia—obscene gestures, self-injury, and vocal tics—which can range from simple throat-clearing to vocalizing obscene words (coprolalia). Tics usually are suppressed during sleep and become more prominent during times of stress. Most tic disorders in children are transient and do not interfere with daily activities. About 10% of adults and children experience a tic that persists for at least a month. Tics most commonly affect school-aged males.

### Tourette Syndrome

Tourette syndrome is the most severe of the tic disorders. It usually starts in early childhood with simple motor tics, such as eye blinking or twitching of the face. From there, the character of the tic can vary—from touching, squatting, and twirling to development of vocal tics after a year or two. The vocal tics begin as simple sounds, but

**Quick Quiz**

- At what age do most children stop sucking their thumbs?
- True or false? You should evaluate for other problems an adolescent female who sucks her thumb.
- Do boys or girls > 10 years of age more commonly bite their fingernails?
- Differentiate early- from late-onset trichotillomania.
- True or false? Tics usually are suppressed during sleep.
- A child with ADHD is placed on stimulant medication. What is a possible side effect of this medication?
- You are told that a 4-year-old child is masturbating in the bathtub. What reactions or punishments are appropriate for this behavior?

frequently progress to barks, sniffs, echolalia, or coprolalia. There is an association between this syndrome and obsessive thoughts and compulsions. ADHD and OCD also commonly display vocal tics.

Stimulant medication used to treat ADHD may unmask the tic disorder; stop the medication if the tic becomes more of a problem than the ADHD. Approximately 15–30% of children who are treated with stimulant medications develop motor tics, most of which are transient. In children who have chronic tics or Tourette syndrome (approximately 50–60% of whom have comorbid ADHD), low-to-moderate doses of methylphenidate often improve attention and behavior without worsening tics. On the other hand, withdrawal of chronic methylphenidate in children with ADHD and Tourette syndrome can result in a decrease in frequency and severity of tics, with an increase when methylphenidate is reinitiated. A metaanalysis of studies evaluating treatment of ADHD in children with comorbid tic disorders found that dextroamphetamine prescribed at higher than the usual recommended doses was associated with exacerbation of tics, but methylphenidate was not. Although predicting the effect of medication on tics is not possible, most children with tics and ADHD benefit from moderate doses of stimulants without worsening of tics.

Age of onset on average is ~ 7 years, with males predominating. Those in late adolescence and early adulthood usually see a period of less frequency and intensity of the tics. Tourette syndrome is familial and suggests an autosomal dominant inheritance pattern.

Treatment should be considered when tics disrupt social interactions or daily living, or negatively impact school/job performance. Historically, the best treatment of the tics themselves is haloperidol, which relieves symptoms in ~ 80% of patients, but unfortunately has serious side effects (cognitive slowing, dystonic reactions, tardive dyskinesia). Today, many use fluphenazine, pimozide, risperidone, and tetrabenazine instead as first-line therapy. You can also use clonidine, botulinum toxin injection, or behavioral therapy (habit reversal training) in those who cannot tolerate the side effects of haloperidol. Many times, treating an underlying obsessive-compulsive or other disorder will result in resolution or improvement of the tics.

### Transient Tic Disorder

Transient tic disorder, the most common type of tic disorder, occurs in children under age 18 and is defined as lasting at least 4 weeks but less than 12 consecutive months. These tics typically will show up many times a day, on an everyday basis. Most commonly, eye blinking or facial tics occur; sometimes, vocal-transient tics also occur. Boys are more commonly affected, usually between ages 3 and 10. Treatment is generally parental assurance, but diagnosis is really one of exclusion. Obviously, the end point of this disorder is in its transient nature, so you have to wait at least 4 weeks to see if it goes away to make the diagnosis.



## SEXUAL DEVELOPMENT AND PARENTAL CONCERNS

### OVERVIEW

Note: For all of the following touchy issues, we're going with the presumed standard of care, as published by the AAP, major textbooks of Pediatrics, and/or "expert" opinion. For the ABP Board exam, know that these are what you will likely be asked about. In other words, follow the standard guidelines of management and treatment as outlined here. I realize that everyone has their own opinion about these difficult issues. However, put your opinions aside for the exam and answer the way the ABP wants you to. After all, passing the exam is your goal. There are very different views regarding these subjects, based on religion, ethnicity, and culture, but the key is to always remember to put the child first. Keep your primary concerns for the child, and you'll get these types of Board questions right ☺!

### MASTURBATION

Masturbation, or self-stimulation of the genitals, is universal from infancy to adulthood. It peaks at age 4 years and again at adolescence. Vulvovaginitis, tight clothing, or urethral irritation can trigger masturbation. Counsel children to keep this behavior private. In families with religious prohibition, encourage parents to provide simple teaching and redirecting behavior. Avoid negative reactions or punishments. Masturbation with auxiliary objects is very unusual in childhood and should raise concerns about sexual abuse.

Masturbation does not cause any physical or mental difficulties and is common behavior. Parents with conservative beliefs, religious or otherwise, may be upset to find a child masturbating. Encourage parents to avoid use of phrases such as "it will make you go blind" or other invalid threats.

## SEXUAL EXPLORATION

### Overview

Sexual exploration is common in the first 3 years of life. This includes the handling of one's own or a playmate's genitals. Typically, children may participate in "nude parading," showing one's genitals, or observing others as they toilet or bathe. Between ages 3 and 6, exhibitionism is common, but it becomes noticeably uncommon after this.

Clues to abnormalities include: imitation of sexual intercourse, asking to have sexual intercourse, doll play that includes oral, anal, or vaginal penetration, or putting one's mouth on another's genitals. These behaviors are red flags and should alert you to either sexual abuse or the possibility that the child has witnessed numerous sexual acts. Usually, children do not adopt these behaviors from television or movies alone; but, rather, they have witnessed them personally.

Children with behavioral problems (in particular bipolar or mood disorders) also are more likely to act out in a sexual way. Additionally, children with developmental delay will generally follow their developmental age rather than their chronologic age; so, a grade-school-age child with developmental delay may still be preoccupied with genital touching of themselves or others.

Refer adolescents who repeatedly perform exhibitionistic behavior to a mental health specialist. The "consistent" exhibitionist will usually surface at around age 15.

### Anatomically Correct Drawings

Children ages 3½–5 years will frequently include genitals in their drawings. After age 4 years, the drawings become more gender-specific, especially with regard to clothing and hair on the drawings. It is unusual for older, grade-school-age children to draw genitals on figures; this requires further inquiry if and when it occurs. Consider sexual abuse when an older child includes genitals in a drawing, but know that such a drawing is not diagnostic for abuse.

### Interest in Pornography

Older children and adolescents frequently show interest in pornography. Today, especially with Internet availability, it is quite easy for minors to have intentional or unintentional access. A couple of episodes of "exploring" pornography are normal, but if it becomes persistent or is associated with other abnormal behaviors, pursue further investigation.

Children's exposure to sexually explicit movies and videos has the potential for harm. For the most part, experts agree that the separation of sex from genuine affection and commitment and erroneous or incomplete depiction of the consequences of certain sexual activities are the wrong messages for teenagers. Parents must stay involved in their children's viewing choices across all media—and have open discussions about limits and acceptable content. This might also be an appropriate time for parents to discuss the potential dangers of "meeting" new friends on the Internet and social networking Web sites. Sexual interest is a developmental issue, but acceptable norms are a family driven issue. The AAP supports family-defined norms!

### Cross-dressing

Cross-dressing (the dressing up in the outer clothes of the opposite sex) is entirely normal for children < 5 years of age. Use of the opposite sex's undergarments, however, by a kindergartner or older child is abnormal. Older children and adolescents who experience sexual pleasure from cross-dressing—known as transvestitism—do so with great guilt and stress. For the most part, these kids are heterosexual, but some may be homosexual. Some recommend behavioral modification techniques for the child who cross-dresses, if the child is motivated and if there are no other underlying psychological problems. Do not encourage punishment for cross-dressing due to any etiology, but conduct further investigation to determine the overall well-being of the child.

### Gender Identity and Behaviors

The term "sissy" is used to describe ~ 5% of boys, and "tomboy" ~ 10% of girls. For the most part, this is just a normal variation, but it may be associated with gender identity disorder, atypical sexual orientation, or other sexual issues.

Gender-identity disorder (GID) applies to children who truly believe they are the opposite of their genetic sex. It occurs in about 1/25,000 males and 1/125,000 females. It presents clinically between ages 2½ and 5. These kids are distressed about every aspect of their gender, including their genitals, clothes, sports, and friends. Girls with GID will claim they are growing penises and refuse to sit down to urinate. Boys with GID claim their penises will disappear, and they will grow up to be a woman.

Evidence suggests that GID is not due to behavior of the parents, although there is a higher incidence of parental psychopathology and depression in families of affected children. These children are usually unhappy. Affected boys avoid all sports and many other male-associated activities. The boys are often shy and anxious. About 65% have separation anxiety disorder. Girls tend to participate in boy-type activities.

**QuickQuiz**

- A 5-year-old girl shows her mother how her two dolls "Barb" and "Kyle" kiss each other. The mother is upset that the child shows "Barb" kissing "Kyle's" genital area. True or false? This is normal behavior for a 5-year-old.
- What is gender-identity disorder?
- True or false? Most "tomboys" (aggressive-acting girls) have gender-identity disorder.
- True or false? Homosexual youth make up a large percentage of youth who are homeless.
- Differentiate a migraine from a tension headache.

Most effeminate boys and most tomboys do not have GID. Usually, these groups like being their sex and have no desire to change it. This is very different from the child with GID, who literally abhors his/her own sex and wants to desperately change to the opposite sex.

Current evidence indicates that therapy will not change children with GID. About 75% will be homosexual, bisexual, or have "indeterminate" sexual orientation. Note: Very few homosexuals and lesbians have GID. They like being males and females, but are attracted to others of the same sex.

Note also that some adolescents and adults continue to get sexual pleasure from cross-dressing, but do not have GID. These adolescents/adults have transvestitism only—they wish to remain their sex, but like to dress up in the clothing of the opposite sex.

### Gay and Lesbian Parents

It is estimated that 10–14 million children in the U.S. have a homosexual parent. No data indicate that having a homosexual parent results in an abnormal effect on social functioning or psychological well-being—or increases the risk of homosexuality or gender identity problems—for their children. Data do show that two parents, no matter what the gender(s), improve the child's developmental outcome over one parent. The key is likely the consistency of love and affection for the child.

### Homosexuality

Homosexuality is defined as having persistent same-sex arousal, with a minimal or absent opposite-sex arousal. Prevalence reports vary, but many suggest that 1–10% of men and 1–6% of women are homosexual. Adolescents frequently engage in activities with or fantasize about the same sex, but these isolated events do not indicate a sustained homosexual predilection. Biological and genetic factors are gaining prominence as the etiology for homosexuality. Homosexuality, per se, most likely is not a "choice"; but sexual behaviors and lifestyles are choices all people make, based on their sexual orientations.

Many children become aware of their homosexuality during adolescence and struggle with "coming out." The accompanying issues of guilt and self-doubt lead to academic problems, truancy, peer problems, potential parental rejection, and homelessness. Physical attacks against adolescent males who have "come out" are well documented in the media. The homelessness issue cannot be overstressed: 25–40% of homeless youth are homosexual. Males are more at risk to be homeless than females. Eating disorders are prominent in males. Therapy to "convert" the homosexual into a heterosexual is contraindicated. The key is to encourage parents to be supportive and to persuade the individuals to accept themselves, be responsible for their actions, and develop a strong life plan in line with their future goals and objectives.

## COMMON PAIN SYNDROMES OF CHILDHOOD

## HEADACHE

### Overview

Headaches are the most common recurrent pain syndrome in children. Headaches can be divided into 3 main groups: 1) migraine, 2) tension, and 3) organic. Tension headaches are the most common, followed by migraines. The average age of onset for headaches is ~ 7 years of age. The F:M ratio changes with increasing age:

- < 7 years of age—males are more likely to have headaches
- 7–11 year olds — females = males
- > 11 years of age — females more commonly have headaches

### Migraines

Migraines were once thought to be due to vascular disturbances, but recent data suggest that they may actually be due to abnormalities with serotonin and other brain receptors. To meet the adult definition, migraines must occur at least 5 times, with each episode lasting 1–72 hours without an identifiable etiology.

Two of the following criteria are mandatory to diagnose migraine headaches:

- Pain on one side (although children with migraine can have bifrontal or bitemporal pain)
- Pulsating/throbbing character
- Moderate-to-severe intensity
- Increasing severity with activity

Additionally, the pain is associated with nausea, vomiting, photophobia, and/or phonophobia. Migraines can be spontaneous or induced by psychological stress, menses, certain foods or smells, or other factors. Some migraines are associated with an aura; e.g., flashes of light, zigzag lines, scotoma. Family history is often positive for migraine headaches. More specific information on migraines is in the Neurology section.

## Tension Headaches

Tension headaches are due to muscle contraction and tend to become more severe as the day goes on. They present as a pressing, dull, persistent tightness—often described as a band around the head. Pain is generally bilateral and less intense (when compared to a migraine) and may resolve within 30–60 minutes or persist for several days.

## Organic Headaches

Organic headaches are due to structural abnormalities, metabolic diseases, or infectious etiologies. Severity and frequency usually increase, and they are not relieved with mild, over-the-counter pain relievers. Treatment should involve addressing the underlying problem if possible. Further physical examination, laboratory, and/or radiologic studies are critical to revealing the etiology (Table 2-5). Headaches due to tumors commonly occur in the morning on awakening and are often associated with vomiting, which may offer temporary improvement of pain. For the Boards, remember that pseudotumor cerebri, which often presents with signs and symptoms that suggest an organic headache, may be associated with doxycycline (headache in an adolescent being treated for acne) and hypervitaminosis A (headache in an adolescent taking multiple vitamin preparations or being treated with vitamin A analogs).

**Table 2-5:** Headache Warning Signs

| When to worry about a space-occupying lesion: |
|---|
| • Sleep-related headache |
| • Absence of family history of migraine |
| • Vomiting |
| • Absence of visual symptoms |
| • Confusion |
| • Abnormal neurologic examination |
| **Additional warning signs:** |
| • Growth abnormalities |
| • Nocturnal awakening from headache |
| • Nuchal rigidity |
| • Headache worsened by cough, micturition, or defecation |
| • Recurrent, localized headache |
| • Persistent vomiting |
| • Progressive increase in headache frequency or severity |
| • Lack of response to medical therapy |
| • Known risk factor for intracranial pathology (e.g., neurocutaneous syndrome, macrocephaly, hormonal abnormalities) |
| • Lethargy |
| • Personality change |
| • Pulsatile tinnitus |

## Cluster Headaches

Cluster headache is a distinct syndrome that frequently responds to treatment with oxygen. The term "cluster" is derived from the periodicity of the headaches: They can occur up to several times per day for a few weeks before remitting. The daily attacks may occur at the same hour each day (in 50% of patients). The pain is unilateral, severe (described as an "ice-pick" or "hot poker") and is peri- or retro-orbital. It peaks quickly in 5–10 minutes and resolves in an hour or two. In about 50%, these headaches predictably occur within 2 hours of falling asleep. Cluster headaches are rare in children before the age of 10 years of age, but become increasingly common between the ages of 10 and 20 years.

These headaches are associated with ipsilateral lacrimation, eye redness, and nasal congestion—features which help differentiate the headache from a migraine.

Cluster patients also tend to be restless during attacks, as opposed to most migraine sufferers who prefer a dark, quiet room and stillness. Boys are affected much more than girls (3 to 4:1).

Treatment: The best acute treatment is oxygen. Inhalation of oxygen at 6 L/min x 15 minutes is usually rapidly abortive, acting to inhibit neuronal activation in the trigeminocervical complex. Triptans, including the intranasal spray, may also be effective. For patients who don't get better with $O_2$ and can't take triptans, octreotide, intranasal lidocaine, and ergot drugs are options.

Once a patient experiences the first of what will become a cluster headache, prophylactic treatment can be instituted. Verapamil is the drug of choice. Other agents sometimes used include lithium, methylsergide, prednisone, and topiramate. The corticosteroids are used as acute drugs while waiting for verapamil to work. Taper off the meds once the cluster is over.

## Chronic Headaches

Chronic daily headache (CDH) is defined as headache that is present for more than 15 days a month for more than three months in the absence of detectable organic

**QuickQuiz**

- True or false? Abdominal pain that is nonorganic in nature will present with growth and development problems in the child.
- Are growing pains actually due to "growing"?

pathology. CDH encompasses 4 subtypes of daily headache defined by the International Headache Society (IHS):

1) Chronic migraine
2) Chronic tension-type headache
3) New daily persistent headache
4) Hemicrania continua

In children aged 12 to 14 years, the overall prevalence is ~ 1.5% and is more common in girls than boys. Most of the adolescents with CDH had chronic tension-type headache or chronic migraine (66% and 7%, respectively) by IHS criteria. Medication overuse has been reported in 20–36% of adolescents with daily headache and is an independent predictor of CDH persistence. Major depression is another independent predictor.

## ABDOMINAL PAIN

Abdominal pain is a common complaint in childhood. Peak incidences are at ~ 7–10 years of age, and these occur more commonly in girls than boys. ~ 35% of children will report abdominal pain lasting 2 weeks or longer.

Most define recurrent abdominal pain as occurring at least 3 times in 3 or more months. It can be dull, crampy, or sharp in character and usually involves the periumbilical area. It is not associated with eating or defecation. The pain may affect daily living, but growth and development are normal—the latter is the key! Environmental stress (e.g., school, sports, social, family); a relative with an ulcer; temperament of the child (e.g., anxious, a perfectionist, lack of coping skills, disordered peer relationships); and secondary gain from having the abdominal pain (e.g., extra attention, getting out of school early) can all influence the rapidity and duration of the pain.

Note: Only ~ 5% of children with recurrent abdominal pain have a true organic etiology. These include constipation, cholelithiasis, inflammatory bowel disease, celiac sprue, pregnancy, UTI, and lactase deficiency. Because 95% of cases do not involve an organic etiology, it is often difficult to differentiate recurrent benign abdominal pain from, say, irritable bowel syndrome (IBS). Children with IBS usually have a history of a change in stool frequency and/or consistency, relief of pain following defecation, and a history of alternating diarrhea and constipation. It is important in the initial evaluation of recurrent abdominal pain to consider the nonorganic etiologies so they can be explored simultaneously with the organic etiologies. The likelihood of an organic etiology is increased among patients with a history of vomiting (especially if bilious), intercurrent fever, weight loss, bloody stools, pain location away from the umbilicus, and pain that awakens the child from sleep.

## LIMB PAIN

Limb pain, also known as "growing pains," is the most common musculoskeletal problem of children. Anywhere from 10–20% of children have it during the school-age years. The most common peak is ages 7–12 years, with girls twice as likely as boys to be affected.

"Growing pains" are benign limb pains that are not due to growing. Usually, they are bilateral, and the child describes them as a deep, aching pain in the muscles of the legs. Most of the pain occurs late in the day or in the middle of the night and resolves by morning. There is no joint involvement, and no inflammation is present. Always address possible organic etiologies before making the diagnosis of benign limb pain.

Specific etiologies to exclude or consider:

- Trauma (stress fracture, myositis ossificans)
- Bone problems (chondromalacia, Osgood-Schlatter disease)
- Collagen vascular disease (JRA, fibromyalgia)
- Infections (osteomyelitis, septic arthritis, toxic synovitis)
- Cancers (osteosarcoma, Ewing sarcoma)
- Endocrine disorders
- Some storage diseases
- Pediatric fibromyalgia
- Neurosensory pain signaling problem (benign)
- Reflex sympathetic dystrophy

Psychosomatic pain also can be similar to "growing pains," and may be due to school phobia or conversion reactions.

For the Boards, remember the following specific lesions/diseases.

Osteoid osteoma often presents with severe nighttime pain that responds to salicylates and nonsteroidal antiinflammatory agents—but not acetaminophen. Osteoid osteoma is a benign lesion that produces prostaglandins; the proximal femur is the most common location followed by the tibia. Plain radiographs reveal a sharp round or oval lesion < 2 cm in diameter with a homogeneous dense center and a 1–2 mm peripheral radiolucent zone. Don't be fooled into thinking this represents a neoplastic lesion!


*Image 2-1: Legg-Calvé-Perthes Disease*

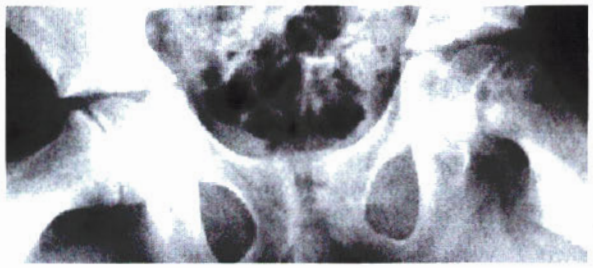


*Image 2-2: Slipped Capital Femoral Epiphysis*

Osgood-Schlatter disease is a repetitive stress injury (often described in a volleyball or basketball player) to the patellar tendon at its insertion into the tibial tubercle. You see it most commonly in children ages 10–15 years. Look for a swollen tibial tubercle. Apophysitis and/or fragmentary ossification of the tibial tubercle may be evident on plain radiograph. Besides mild antiinflammatory agents, no specific therapy is necessary.

Transient synovitis is characterized by pain, limp, and limitation of motion in the hip. The etiology is unclear but may be related to posttraumatic mechanisms or recent upper respiratory infection. Patients appear nontoxic but may refuse to walk—toxicity suggests a septic arthritis! Treatment of transient synovitis is conservative and symptomatic.

Legg-Calvé-Perthes disease is a partial or complete idiopathic avascular necrosis of the femoral head. Look for this in a boy between the ages of 4 and 8 years. It resolves with time and no treatment seems to speed the return of blood flow to the femoral head—make the child non-weightbearing and refer to an orthopedist. See Image 2-1; note the misshapen and "ratty" appearance of the left femoral head.

Slipped capital femoral epiphysis is the slipping of the epiphysis off the metaphysis. Look for an obese, eunuchoid adolescent (more often an African-American), although some patients are thin. Get an AP and frog-leg view. Treat with surgery. See Image 2-2; note the slippage on the left side.

Remember for Boards: Hip pathology often presents with lateral thigh or knee pain!

## CHEST PAIN

Chest pain is very common and is the second most common reason for referral to a pediatric cardiologist. (Heart murmur is #1.) In children, it is very rare that these pains are cardiac in origin. Most are idiopathic. In children > 13 years of age, which is the average age for presentation, the etiology is usually psychogenic or musculoskeletal (e.g., costochondritis, muscle overuse/strain).

History and physical examination guide the need for further studies. More in the Cardiology section.

# ATTENTION DEFICIT DISORDER (ADD, ADHD)

## OVERVIEW

Attention deficit disorder and attention deficit hyperactivity disorder (ADD, ADHD) are controversial in both diagnosis and treatment. The problem is that presentation variability is so broad. Generally, short attention span, inattentiveness, impulsivity, and overactivity characterize this disorder. Most children without the diagnosis of ADD/ADHD occasionally have these same traits, which adds to the difficulty of defining who has the syndrome and who is just an average, everyday kid.

Approximately 9.5%, or 5.4 million, of children 4–17 years of age have been diagnosed with ADHD, as of 2007. The percentage of children with a parent-reported ADHD diagnosis increased by 22% between 2003 and 2007. Rates of ADHD diagnosis increased an average of 3% per year from 1997 to 2006 and an average of 5.5% per year from 2003 to 2007.

DSM-IV lists behaviors that make up the diagnostic criteria for ADHD. Look for them in Table 2-6. Boys are much more likely to be affected.

**Table 2-6:** Behaviors Suggestive of ADHD

| Inattentive Behaviors |
|---|
| Easily distracted by extraneous stimuli |
| Makes careless mistakes in schoolwork or other activities |
| Has difficulty maintaining attention to task |
| Does not seem to listen to what is being said to them |
| Fails to finish schoolwork, chores, or other duties |
| Loses things necessary for tasks or activities |
| Has difficulty organizing tasks and activities |
| Forgetful in daily activities |
| **Hyperactive / Impulsive Behaviors** |
| Runs about or climbs excessively in inappropriate situations |
| Fidgets with hands or feet or squirms |
| Has difficulty awaiting turn in games or groups |
| Blurts out answers to questions |

**QuickQuiz**

- Describe a typical child with ADHD.
- True or false? Sugars and artificial flavorings increase the risk of developing ADHD.

## ETIOLOGY

For most children, no etiology can be found. Genetic factors are possible in some children; there is an increased frequency of alcoholism, substance abuse, sociopathy, and hysteria among parents of children with ADHD. Toxin exposure both prenatally and postnatally has been implicated. And it is well known that fetal exposure to alcohol, cocaine, or lead may result in attention and hyperactivity problems. Controlled studies have failed to consistently implicate sugars, artificial flavorings, and salicylates as etiologies.

Recent theories postulate that ADHD is actually an inhibitory control problem and/or a difficulty with goal persistence. These theories are still controversial and not widely accepted.

One thing we do know: ADD does not have a single cause.

Evaluation must include a detailed medical, family, and social history and complete physical examination. Family members, other caregivers, and teachers should be asked to complete standardized assessment forms (e.g., Vanderbilt scales). By definition, symptoms should be present in all settings—school, home, day care. Marked differences in symptoms between settings is not consistent with ADD/ADHD but rather suggests, for example, a dysfunctional relationship between parent and child or teacher and child. These scales also assess for associated co-morbidities—depression, anxiety, oppositional behaviors, and/or conduct disorders.

## CLINICAL FINDINGS

The clinical findings center on 5 main areas:

1) Easy distractibility, fails to give close attention to detail or stay on task
2) Impulsivity (e.g., difficulty waiting for turn, blurts out answers, talks excessively)
3) Hyperactivity and inability to sit still (e.g., fidgets, leaves seat, "on-the-go")
4) Inability to organize or plan tasks, often loses things, "forgets" assignments
5) Emotional lability

Usually it is important to note that some hyperactive-impulsive or inattentive symptoms that caused impairment were present before the age of 7 years. Also note that many suggest that some impairment from the symptoms is present in two or more settings (e.g., at school, work, and/or at home).

## LABORATORY

Generally, laboratory testing is unnecessary; but consider lead screening, especially if history dictates—and know that thyroid abnormalities are more common in children with ADHD than in the unaffected population. Routine use of EEG, MRI, etc. is not indicated.

## TREATMENT

Treatment is multifactorial. Behavior management is important and can include positive reinforcement strategies, such as "time-outs," and "extinction" techniques—systematically ignoring undesirable behaviors. Preferential seating in the front of the class and away from windows and doors, assignment calendars with stickers, and frequent communication between teacher and parent should be encouraged. Children with ADHD often have associated learning disabilities, are anxious, and struggle with issues of self-esteem, especially as they grow older.

It is also important to consider comorbid conditions and referral as necessary for these associations:

- Learning disabilities (15–25%)
- Language disorders (30–35%)
- Mood disorders (15–20%)
- Anxiety disorders (20–25%)

Drug therapy is beneficial for a majority of children affected with ADHD. Most commonly used stimulant medications: methylphenidate (available in several different preparations including Concerta®, Ritalin-LA®, Metadate®), dextroamphetamine, and Adderall® (a single entity amphetamine product combining the neutral sulfate salts of dextroamphetamine and amphetamine, with the dextro isomer of amphetamine saccharate and 6, l-amphetamine aspartate). All are now available in short-acting (~ 4 hours) and extended release (~ 6–8 hours) preparations. They all increase the release and inhibit the reuptake of dopamine and norepinephrine. Lisdexamfetamine (Vyvanse®), a newer pro-drug for treatment of ADD/ADHD, has less abuse potential than its active metabolite (dextroamphetamine). It is initially inactive following consumption and slower to metabolize than dextroamphetamine.

Side effects include the potential for growth suppression, weight loss due to appetite suppression, headache, hypertension, abdominal pain, and exacerbation of tic disorders—although recent data/reports suggest that many tics actually improve or do not worsen. Some children and adolescents (and their friends and parents) complain of irritability and feeling "out of sorts" as the "medication wears off."

A common Board question may ask ways to monitor the child on medication(s) for ADHD. These include monitoring blood pressure, weight, and appetite; being aware of increased risk of sleep disturbance and tics; counseling the child on the risks of concomitant use of substances of abuse; and the likelihood others might encourage misuse of the medication, including of giving/selling the medication to someone else.

In 2006, the FDA advisory committee initially recommended a "black box" warning on ADHD stimulant medications because of the perceived increase in adverse cardiovascular events. However, data failed to show that ADHD stimulant medications actually increased the risk of sudden death and no "black box" warning was required. But a "black box" warning was added that says "misuse of amphetamines may cause sudden death and serious cardiovascular adverse events." The AAP does not recommend screening ECGs unless the patient's history, family history, or the physical examination raises concerns. (Note that the American Heart Association did recommend ECGs, but the AAP did not feel they were necessary because serious cardiovascular events are very rare in children taking stimulant medications and was equal to the rate in the general public.) Atomoxetine (Strattera®) is a selective norepinephrine reuptake inhibitor. It is the only ADHD med that is not a controlled substance and its side effects include liver injury, suicidal thoughts, tics, and weight loss. It is approved for use in children > 6 years of age. Other agents include guanfacine (now also available in a long-acting daily preparation, Intuniv®) and clonidine, both of which are helpful for those with aggressive or hyperaroused behavior and in patients unable to tolerate stimulant therapy.

Non-conventional treatments, such as mineral therapy, diet restriction, and other dietary manipulations have failed to show improvement in controlled trials.

## PROGNOSIS

Children with ADD/ADHD may persist into adulthood with the disorder, although the hyperactivity symptoms tend to diminish over time. Untreated children and adolescents with ADHD have an increased risk of intentional and unintentional injuries; those who drive have an increased risk of motor vehicle accidents! Pharmacologic treatment of ADHD is associated with a decreased risk of substance abuse during adolescence.

# DISRUPTIVE BEHAVIORS

## OPPOSITIONAL DEFIANT DISORDER

Oppositional defiant disorder occurs in otherwise average children during the school-age and adolescent stages of development. Boys are much more commonly affected than girls. Key to the behaviors is a duration of at least 6 months. Table 2-7 summarizes DSM-IV diagnostic criteria.

## CONDUCT DISORDER

Conduct disorder is a complex, multifactorial disorder. Generally, think of it in kids who have repetitive, persistent (> 6 months) behaviors that violate the rights or property of others. These children lack guilt or remorse about their behavior. These are the kids who have been caught repeatedly stealing, lying, fighting, harming animals, setting fires, using drugs, or instigating sexual abuse.

There are 2 groups in conduct disorder:

1) The undersocialized group comprises kids who are unpopular, lack any close friends, and tend to be socially isolated. They lack any empathy for their age group and despise adults. The behaviors occur at home, at school, and in the community.
2) The socialized group participates with a peer group in their behaviors—they have strong interpersonal ties but are confrontational with adults.

Refer children and adolescents with conduct disorder for behavioral and psychotherapeutic intervention.

# PERVASIVE DEVELOPMENTAL DISORDERS

## OVERVIEW

Pervasive developmental disorders comprise a wide group and occur at a rate of ~ 5–7/100,000. Males are much more commonly affected, except with the incidence of Rett syndrome. Etiologies for these conditions are unknown, but some evidence indicates that genetics may be a factor. In autism, you will see elevated levels of serotonin in platelets in ~ 33% of affected

**Table 2-7:** Four Criteria for the Diagnosis of Oppositional Defiant Disorder

1. **Behavior has to last at least 6 months, and 4 or more of the following must occur more frequently than is commonly seen for that age group:**
   - **Often loses temper**
   - **Often argues with adults**
   - **Actively defies or refuses to comply with adults' rules/requests**
   - **Annoys people on purpose**
   - **Blames others for his/her misbehavior**
   - **Easily annoyed by other people**
   - **Often angry or resentful**
   - **Often spiteful or vindictive**
2. **The behavior must cause clinically significant deviation in social, academic, or occupational functioning.**
3. **The behaviors do not always occur in association with a psychotic or mood disorder.**
4. **Criteria are not met for conduct disorder, and, if older than 18 years of age, subjects don't meet criteria for antisocial personality disorder.**

**QuickQuiz**

- How long must a behavior occur for it to be classified as oppositional defiant behavior?
- Do children with conduct disorder have guilt or remorse over their actions?
- Which pervasive developmental disorder is usually seen only in girls?

individuals—although the significance of this finding is unknown. Major characteristics of patients with this disorder include impaired social interaction and communication—often associated with stereotypic behaviors, interests, and activities. Affected individuals are often extraordinarily knowledgeable about a particular topic (e.g., trains). Generally, prognosis is poor among most children with these disorders, and assistance from other health care providers (psychologists, social workers, therapists, etc.) will be needed.

## AUTISM

Autism is now a subset of "pervasive developmental disorders" and is the most common one.

There are multiple diagnostic criteria for autism that spread over 3 areas:

1) Social interactions
2) Verbal and nonverbal communications
3) Behaviors and activities (symbolic and imaginative play)

To meet the definition, the developmental problems of autism must begin < 3 years of age and must exist in more than one setting. AAP recommended routine screening is discussed in the Growth and Development / Preventive Pediatrics section. Diagnosis is suggested by the criteria in Table 2-8: DSM-IV Criteria for diagnosis of Autistic Disorder.

## RETT SYNDROME

Children with Rett syndrome have many of the features of a child with autism. With Rett syndrome, prenatal, perinatal, and early development up until age 5 months is normal; but then, head circumference growth dramatically declines followed by rapid developmental deterioration, respiratory dysfunction, severe psychomotor retardation, and loss of limb function leading to wheelchair dependence. One of the first signs is deceleration of head growth followed by symptoms such as seizures, ataxia, and autistic features. Stereotypic hand wringing is almost always associated. It occurs almost exclusively in girls and is due to a mutation in the *MECP2* gene.

## ASPERGER SYNDROME

Asperger syndrome is similar to autism in that these children have severe and pervasive impairment in social interactions and also have restrictive, repetitive, stereotypic behaviors. They often have obsessional

**Table 2-8:** Autistic Disorder: Criteria for Diagnosis

A. A total of 6 (or more) items from (1), (2), and (3), with at least 2 from (1) and 1 each from (2) and (3):

1. Impairment in social interaction:
   a. Marked impairment in the use of multiple nonverbal behaviors (e.g., eye-to-eye gaze, facial expression, body postures)
   b. Failure to develop peer relationships on par with developmental level
   c. Lack of spontaneous seeking to share enjoyment, interests, or achievements with others (e.g., by a lack of showing, bringing, or pointing out objects of interest)
   d. Lack of social or emotional reciprocity

2. Impairments in communication:
   a. Delay in, or total lack of, development of spoken language, with attempt to compensate through alternative modes of communication, (e.g. pointing, gesture or mime)
   b. In individuals with adequate speech, impairment in the ability to initiate or sustain a conversation with others
   c. Stereotyped and repetitive use of language or idiosyncratic language
   d. Absence of spontaneous make-believe play or social imitative play on par with developmental level

3. Restricted, repetitive, and stereotyped patterns of behavior, interests, and activities:
   a. Preoccupation with ≥ 1 stereotyped and restricted pattern of interest that is abnormal in either intensity or focus
   b. Inflexible adherence to specific, nonfunctional routines or rituals
   c. Stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting or complex whole body movements)
   d. Persistent preoccupation with parts of objects

B. Delayed or abnormal functioning in at least 1 of the following areas, with onset < 3 years of age:
   1. social interaction, 2. language as used in social communication, or 3. symbolic or imaginative play

C. Clinical signs and symptoms are not better accounted for by Rett disorder or childhood disintegrative disorder.

and idiosyncratic interests (e.g., vacuum cleaners, calendars, business cards) that they are able to describe in great detail and with broad knowledge of the subject. The difference, compared to autism, is that these children develop normal language and cognitive development skills.

# PSYCHIATRIC DISORDERS

## OBSESSIVE-COMPULSIVE DISORDER

Obsessive-compulsive disorder is a complex syndrome that may border on "normal areas" at some point. Generally, consider OCD when rituals or superstitions lose their age appropriateness, cause excessive actions (hand washing, etc.) associated with marked anxiety and distress, impede socialization, or detract from normal duties and experience. Obsessions must be persistent and recurrent and involve senseless ideas, images, or impulses that intrude on normal activities. The obsessions must be involuntary. Compulsions are repetitive, purposeful behaviors that are performed by following "rules" that must be applied rigidly, in response to an obsession, and are often done in an attempt to stop the obsession with the hope of preventing some dreaded event. Those affected do not deem the compulsions as pleasurable, but believe them to be necessities. An example: the compulsion to check the alarm clock every 15 minutes to be sure it is set.

Recent data support OCD as a neurobiological disorder; there also is an increased familial nature to the disorder. Boys are more commonly affected and tend to have an earlier age of onset than girls.

The most common OCD "ritual" in childhood is repetitive cleaning and washing. This includes showering, hand washing, and toothbrushing. Other common OCD rituals include going in and out of doors; rereading; rechecking doors to be sure they are locked; rechecking stove and appliances to be sure they are off; rechecking the alarm clock; and counting, ordering, and arranging objects. However, the most common obsessions in children concern dirt and germs. Other obsessions include the feeling that something hasn't been done correctly or that something terrible is going to happen.

The differential for OCD is extensive. Tourette syndrome can involve complex tics that can resemble the rituals of OCD. Excessive rumination on gloomy thoughts is a common feature of depression; but the depressed person views these thoughts as making sense while the person with OCD views them as abnormal. Note: Eating disorders, by definition, are excluded from the diagnosis of OCD.

Boards: Remember that some children experience worsening of OCD symptoms (and/or tics) concurrent with an infection due to group A beta-hemolytic streptococci—AKA: Pediatric Autoimmune Neuropsychiatric Disorder Associated with Streptococci, or PANDAS, in which antibodies to group A streptococcus are proposed to cross-react with the basal ganglia resulting in symptoms.

Also remember that almost all children experience some superstitions or rituals at age-appropriate levels. For example, children may collect Pokémon cards, elaborately store them, and categorize them. For most kids, this is an enjoyable activity and provides them time to socialize with friends who have similar interests. In contrast, a child who collects the cards but keeps them wrapped in plastic to prevent germs from getting on them, or refuses to let friends see or handle the cards and who does not seem to have any enjoyment in sharing or discussing the cards with another child, actually may be exhibiting OCD-type behavior. Remember: The OCD behavior is usually distressing to the affected child as well.

Direct treatment for OCD behavior is expressive and supportive psychotherapy. Behavioral therapies are also helpful, as are pharmacologic therapies, such as serotonin reuptake inhibitors (SSRIs), although recent overuse has brought these drugs into question. Any of the agents work, including fluoxetine, sertraline, fluvoxamine, and paroxetine. Clomipramine, a tricyclic, may also be helpful.

Studies show that ~ 50% of children with OCD continue to have OCD as adults.

## MÜNCHHAUSEN SYNDROME BY PROXY

Münchhausen syndrome by proxy is a bizarre disorder in which the caregiver-child relationship is "disturbed." The mother, almost always the one involved, is deliberately dishonest about a history of illness in her child and/or harms the child to create an illness. Usually the mother has a psychiatric illness. These actions against the child are considered a form of child abuse. Children may be at risk at any age, but this most commonly affects infants and school-aged children. Frequently, no father is present, and the mother is socially isolated, appears to be extremely devoted to her child, and is very knowledgeable about the child's "illness."

Interestingly, the mother is frequently a member of the health care profession.

Clues that Münchhausen syndrome by proxy may be occurring:

- Recurrent, serious illness that cannot be satisfactorily explained.
- Failure of routine management of an illness.
- Symptoms do not match with scientific evidence.
- History of illnesses in the past is inconsistent.
- History of an unexplained illness or death of a sibling.
- A mother who appears inappropriately calm or even euphoric when her child is in the hospital.
- A mother who seems inappropriately excited that her child is going to be admitted.

**Quick Quiz**

- How does Asperger syndrome differ from autism?
- Define the terms "obsession" and "compulsion" as they relate to obsessive-compulsive disorder.
- What is the most common OCD "ritual" in childhood?
- What is the most common obsession in children?
- Describe the usual perpetrator in Münchhausen syndrome by proxy.

- You get a creepy feeling just being around the mother.
- She requests specific diagnostic studies or procedures of the child.

This disorder is difficult to sort out, because many inborn errors of metabolism or unusual diseases may "not respond to routine management" or may not "be satisfactorily explained." In the real world, be sure you have ruled out just about everything else before you consider this diagnosis. Diagnosis is very tricky and involves a multidisciplinary approach, including referral to mental health workers familiar with this disorder. Court-ordered video surveillance during hospitalization has oftentimes provided evidence that the "cause" of the child's symptoms is due to his mother's actions; e.g., a parent is filmed using a lancet to cut her finger and then deposits the blood in her child's diaper. Long-term prognosis and outcomes are guarded and usually not good for these families. Usually, on the Board examination, you can pick these questions out because the mother's history notes she is a nurse or physician or other health care professional who obsesses about medical details (clue: why are they telling me about the parents profession?) and you begin to get a creepy feeling (2nd clue) while reading the question that something is "just not right."

Note: Depression is covered in the Adolescent Health and Gynecology section.

## OBESITY

Obesity is defined as having a body mass index (BMI) $\geq$ the 95th percentile. "Overweight" is defined as having a BMI $\geq$ 85th and < 95th percentile. Severe obesity is $\geq$ BMI of 35. A strong risk factor for obesity in a child is having an obese parent (no difference which one), and there is even more risk if both parents are obese. If you are obese at age 10–14 years and you have at least 1 obese parent, the risk of being an obese adult is 80%. In 2007–2008, nearly 1/3 children and adolescents were overweight or obese!

Recently, we discovered "obesity genes," but there is still considerable controversy about whether they are completely responsible for the condition. Most likely, heredity plays a role in the susceptibility of a person to become obese; however, it is the behavior (e.g., lack of exercise, sedentary activities in front of the television or computer) and environment (e.g., easily accessible high-caloric fast foods) of the individual that determines if the obesity will be phenotypically expressed.

We know there are 3 critical periods in childhood that result in an increased likelihood of persistent obesity:

1) Prenatal period
2) Age 5–7 years
3) Early adolescent years

The more common problems with adolescent obesity include dyslipidemia, hyperinsulinemia, and a markedly increased risk of obesity in adulthood. Obese adults are at increased risk for complications from diabetes, hypertension, heart disease, cancer, joint problems, and digestive disorders.

Treatment of obesity must be family- and behavior-oriented. Family support and involvement are critical. Concurrent changes in dietary (e.g., portion sizes) and patterns of physical activity (e.g., turn off the television and get outside) are most often associated with a successful outcome. Parents must be invested (better yet, involved themselves) in the program if weight loss is to occur. Results are still disappointing but better than those seen in adult programs. About 35% of children who actively participate in a state-of-the-art program will be obesity-free at 10 years. The family and the child both must be ready and willing for intervention to succeed.

## METABOLIC SYNDROME

The metabolic syndrome is not a disease itself but is a cluster of related diseases that are well described in adults and associated with increased cardiovascular and other health risks. As of this book's publication, as well as according to the 2008 Bright Futures Guideline, a "definite" definition has not been accepted for children. Table 2-9 lists criteria for metabolic syndrome in children and adolescents. Most propose that having 3 or more measures in the table should make the diagnosis of metabolic syndrome. More on this in the Endocrinology section.

Table 2-9: Suggested Metabolic Syndrome Indices in Children and Adolescents

| |
|---|
| BMI: > 97th percentile |
| Triglycerides: > 110 mg/dL |
| HDL cholesterol: < 40 mg/dL |
| Systolic/diastolic BP: > 90th percentile |
| Glucose abnormalities: Fasting glucose > 110 mg/dL or oral glucose tolerance test > 140 mg/dL |
| Waist circumference: > 90th percentile |

MedStudy

PEDIATRICS BOARD REVIEW

PEDS

**CORE CURRICULUM**

5th EDITION

Authored by J. Thomas Cross, Jr., MD, MPH, FAAP
with Robert A. Hannaman, MD

# EMERGENCY PEDIATRIC CARE



Many thanks to the Emergency Pediatric Care Advisor:

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# Emergency Pediatric Care

# CHILD MALTREATMENT SYNDROMES

## OVERVIEW

Information here is presented from the 2009 report from the National Child Abuse and Neglect Data System. Child maltreatment syndromes reported include neglect (78%), physical abuse (18%), sexual abuse (10%), and psychological maltreatment (8%). In 2009, over 3.6 million reports were made to child protective services (CPS), and ~ 710,000 children were determined to be abused or neglected (~ 20% of reports are substantiated). The highest rate was in the age range of birth to 1-year-old. Boy and girls had nearly identical rates of abuse, 48% versus 51%. However, genders are not equally affected: Boys are more likely than girls to be physically abused, but 75% of sexual abuse cases involve females. In fact, it is estimated that 1 in 4 girls and 1 in 10 boys have been sexually abused.

Although clinicians must maintain a high level of suspicion whenever seeing an injured child, there are characteristics of children, caretakers, and families that are associated with maltreatment. Characteristics of abused children include those that interfere with bonding (e.g., prematurity, neonatal separation, multiple birth), as well as congenital defects, retardation, and provocative behavior. Abusive caretakers may themselves have been abused/neglected, be young parents, or have problems with mental illness or substance abuse. Abusive families often live with financial stress, which often is associated with being in a racial minority. Sexual perpetrators are almost always male, known to the abused child, and about 20% are < 18 years old.

For all types of abuse, it is paramount that communication exists between the parents and the clinician. But of utmost concern is the safety and well-being of the child. Generally, child protective agencies are available to assist the clinician. But frequently, it may fall to you to provide relevant information to designated authorities—or to actually decide on whether it is appropriate for the child to return to the home environment or if the child requires alternate shelter, including possibly hospitalization.

## PHYSICAL ABUSE

The classic definition of physical abuse is "an act against a child that results in harm or intended harm to the child." Many limit this to "deformation or leaving a lasting mark on the child's body." This definition tries to separate physical abuse from common practices, such as "spanking." But the difference between these definitions remains blurry. For the Boards, think of physical abuse as beating, shaking, scalding, or biting.

The following should raise your suspicion for physical abuse:

1) The history of how the child sustained the current injury is not known, not appropriate for the child's age (e.g., a 1-month-old can't roll off a bed), not compatible with the injury, changes from one telling to the next, or is different from family member to family member.
2) Delay in seeking medical care for a significant injury.
3) History of recurrent injuries, especially those with inconsistent explanations.
4) History of abused sibling or unexplained death of a sibling.

Certain findings on physical exam also trigger suspicion. For example, consider developmental characteristics. A 1-year-old just learning to walk will frequently have bruises on the face or shins from falling, but it is unusual to see bruising on a child who is not yet cruising or crawling.

The most commonly reported:

- Form of child abuse: neglect
- Manifestation of physical abuse: soft tissue injury
- Cause of death due to physical abuse: head injury
- Fractures seen in child abuse: skull fractures, specifically linear fracture of the parietal bone

Soft tissue injuries (hand marks from slapping, bruises, linear marks from belts or cords, and bite or burn marks) raise suspicion if in an area not harmed with normal falls (e.g., black eyes, around the ears, genital area, posterior body surfaces) or if not consistent with the story (e.g., "she fell forward and bruised herself"—hmm, how did the back of her leg get bruised falling forward?).

Estimate the bruise's age by color:

| | | |
|---|---|---|
| • < 1 day | Red | (red to reddish blue) |
| • Day 1–4 | Blue | (dark blue to purple) |
| • Day 5–7 | Green | (green to yellow green) |
| • Day 7–10 | Yellow | (yellowish to brown) |
| • Week 1–3 | Normal | |

Burns account for 5% of physical abuse. These range from scalds to cigarette burns. Patients with immersion burns present with bilateral burns to the buttocks or back and will usually have a sharp area of demarcation between normal and burned skin, the so-called "stocking glove" distribution. Non-abusive burns will usually be asymmetric (e.g., only one foot due to stepping on a burn source) or otherwise uneven, such as a hot liquid that was accidentally spilled on the child. Multiple cigarette burns are strong evidence for abuse; a single burn may be a sign of abuse if it is in an unlikely area, such as the back, buttocks, or genital area.

Head injury in infants < 1 year is most commonly due to child abuse. Shaken impact or shaken baby syndrome results in subdural or subarachnoid hemorrhages and retinal hemorrhages. These injuries are caused by repeated

accelerations and decelerations of the brain, producing shearing of the bridging vessels and consequent intracranial bleeding. Many shaken babies also have injury due to impact with the floor, crib, etc. Presentations include seizures, coma, apnea, or arrhythmias—or simply, "the baby is not acting right." Frequently, there are other signs of abuse, such as bruising, rib fractures, etc.

Bone fractures also are a common sign of child abuse. Metaphyseal chip fractures are caused when an extremity is yanked and the periosteum—most tightly adherent to metaphysis—causes a small bone fragment to avulse. Consider abuse with fractures of skull, femur, or ribs (especially anterior and posterior rib fractures). Some fractures are suspicious because they are uncommon (vertebrae, sternum, pelvis, scapulae), some because the mechanism could be twisting (spiral fracture of humerus), and some because the patient is too young to walk. Note: It is common for a 2–3-year-old to have a supracondylar humerus fracture from a fall to the elbow, or a spiral tibia fracture from a twisting fall. Spiral fractures by themselves are not diagnostic of abuse. Two fractures more specific for abuse are:

1) Bucket handle (epiphyseal-metaphyseal junction) fracture
2) Rib fractures, particularly those posterior and near the spine (These are not due to CPR!)

Other injuries that raise suspicion of abuse are lacerations to the liver or spleen and suspicious poisonings. Multiple SIDS deaths in a family have been linked to abuse by suffocation. When more than 1 infant in a family dies of SIDS, investigate both child abuse and other causes, such as metabolic diseases.

Münchhausen syndrome by proxy is an extreme form of abuse in which the mother (rarely the father) fabricates or creates illness symptoms in the child and takes pleasure in the child undergoing multiple tests/procedures. Child victims may be made ill by poisoning or injection with feces, for example.

Evaluation: For children with serious head injuries, perform an ophthalmologic exam to look for retinal hemorrhages and order CT or MRI scan. If abuse has occurred in a child < 3 years of age, get a skeletal survey, which includes x-rays of skull, chest, pelvis and lumbar spine, extremities, and hands and feet. To date a fracture, remember that a soft callus forms at 7–14 days. Before this time, there's a sharp fracture line; after this time, there's a hard callus. Bone scans are useful for detecting acute rib fractures and other older fractures. Lab tests include CBC, lead level (child < 6 years), and U/A. If bleeding or bruising is present, PT, PTT, and platelet counts are appropriate. Order liver transaminases if you suspect abdominal trauma.

## NEGLECT

Neglect, the most common form of child abuse, includes "acts of omission," such as failure to provide adequate food, shelter, clothing, or supervision. It includes abandonment and failure to provide a child with adequate health care and/or education.

A common manifestation of neglect is poor growth and developmental delay. Older neglected children may seem "emotionally needy," or they can be very adult-like in their behaviors due to forced early independence and self-reliance.

## SEXUAL ABUSE

Sexual abuse is involvement of a child or adolescent in sexual activities they do not comprehend, to which they are unable to consent, or which violate societal norms. The child usually knows the perpetrator, who is often a relative. Sexual abuse includes rape, statutory rape, indecent assault, incest, and prostitution. Clinical manifestations may be behavioral—either specific (e.g., excessive masturbation, sexually overt behaviors) or nonspecific (e.g., poor school performance, anxiety, suicidal gestures). Similarly, physical complaints may be specific (e.g., genital or rectal laceration, pregnancy) or nonspecific (e.g., anorexia, abdominal pain, dysuria, vaginal discharge). Vaginal discharge alone represents a low risk (< 5% of the time) of indicating sexual abuse.

Few sexually abused children have abnormal genital or anal findings. Acute female genitalia trauma (e.g., abrasions, lacerations, or hematomas) should raise your suspicions about a recent abuse incident. Past abuse may manifest as U- or V-shaped clefts in the posterior hymenal rim or decreased width of hymenal tissue posteriorly. These findings are especially suspicious if they persist when you examine the child in the prone, knee-chest position. An enlarged hymenal opening does not by itself indicate abuse. For males, findings are rare, except for acute anal fissures and thickened rugae.

Obviously, pregnancy or an STD is definitive evidence that sex has occurred. Gonorrhea, *Chlamydia*, and human papillomavirus are the most commonly found STDs.

Most cities and hospitals have protocols for the evaluation of sexual abuse. These include contacting appropriate law enforcement authorities, gathering appropriate forensic evidence, and ordering necessary laboratory tests.

## EMOTIONAL MALTREATMENT

Emotional maltreatment is probably one of the most common forms of abuse, but it is difficult to define or identify. It includes repeated verbal denigration, belittling, "making fun of," and scapegoating for problems that have nothing to do with the child. These attacks result in a child with low self-esteem and feelings of worthlessness.

## QuickQuiz

- Who are more commonly sexually abused—boys or girls?
- A 1-year-old presents with a broken left femur. The mother says the child was with his father for most of the day, while the mother was out shopping. The mother says she found the child in his crib refusing to bear weight on the leg. The mother says she doesn't know how the child was injured. True or false? You should be suspicious of physical abuse.
- A 1-year-old boy presents for an otitis media follow-up visit. The child's left cheek has a dark purple-blue bruise. The father says his son fell against the coffee table edge 2 days ago when attempting to walk. A few scrapes are noted on the child's shins. True or false? You should be suspicious of physical abuse.
- What is the most common cause of death due to child physical abuse?
- What eye finding is important to look for in a child you suspect may have been "shaken" violently?
- True or false? Rib fractures should make you suspect child abuse.
- What radiologic test is useful for detecting acute rib fractures?
- True or false? In a child you suspect has been sexually abused, a normal genital and rectal examination rules out sexual abuse as a possibility.
- What are the common causes of miosis and mydriasis?

# POISONINGS

## OVERVIEW

In the U.S., over 2 million poisoning events occur annually, and over 60% of these occur in children under the age of 6. The peak age for ingestions is 18 months to 3 years. 92% of the events occur in the home and involve only 1 substance. Luckily, 75% can be managed in the home.

85% of all poisonings are unintentional and generally occur in children younger than 5 years of age. Boys are more commonly affected than girls, and > 50% are nonpharmaceutical ingestions—the most common being cosmetics, cleaning products, plants, and foreign bodies. As for the pharmaceuticals, the most common unintentional ingestions are acetaminophen, cough and cold preparations, vitamins, and antibiotics.

Of the 15% of poisonings that are intentional, a majority occur in adolescents and adults. Females are more commonly affected, and pharmaceuticals make up a majority of these cases, including acetaminophen, barbiturates, stimulants, and antidepressants, as well as alcohol.

## INITIAL MANAGEMENT

### Overview

Initial management for all poisoning follows the ABCDs and stabilization management rules: Airway and antidotes, breathing, circulation, and disability/decontamination.

The physical exam hints at what the toxin is. Pay attention to pupil size (miosis = pinpoint, or Dilated = myDriasis).

Causes of miosis include (COPS):

- Cholinergics, clonidine
- Opiates, organophosphates
- Phencyclidine, phenothiazine, pilocarpine
- Sedatives (barbiturates)

Causes of mydriasis include (AAAS):

- Anticholinergics (atropine)
- Antihistamines
- Antidepressants (cyclic)
- Sympathomimetics (amphetamine, cocaine, LSD)

Causes of diaphoretic skin (SOAP):

- Sympathomimetics
- Organophosphates
- Aspirin (salicylates)
- PCP (phencyclidine)

Causes of red skin:

- Carbon monoxide
- Boric acid

Causes of blue skin:

- Cyanosis
- Methemoglobinemia

Other clues include laboratory findings:

- ECG: Look for prolonged QRS in tricyclic ingestions.
- Osmolar gap: Look for in unknown alcohol ingestion.
- Anion gap: Look for in MUDPILES (methanol, uremia, DKA, phenols, iron, INH, lactate, ethanol, ethylene glycol, and salicylates).
- X-rays: These may pick up pill fragments (CHIPES: chloral hydrate, calcium, heavy metals, iron, phenothiazines, enteric-coated preparations, sustained-release tablets).

In the management of poisoning, you'll want to prevent agents from being further absorbed, depending on the type of exposure:

- Dermal: Remove clothing, wash skin with water and then soap and water (1st priority for organophosphate poisoning).
- Ocular: Irrigate eyes with copious (1,000 cc) NS.
- Respiratory: Remove patient to fresh air.
- GI: See the more extensive discussion next.

### Detoxification

Most liquids are absorbed in 30 minutes, and most solids are absorbed in 1–2 hours.

Activated charcoal is used in the first hour to absorb substances and decrease bioavailability. *In vitro*, 10 grams of charcoal will generally absorb 1 gram of toxin. For a child, generally give 1 gram/kg of activated charcoal; for adolescents and adults, give 50–100 grams. It may interrupt enterohepatic circulation of drugs; e.g., salicylates. Complications include pulmonary aspiration, emesis, and constipation. Contraindications include having GI obstruction or perforation, unprotected airway, or ingestion of caustic or hydrocarbon. (Charcoal use now is being scrutinized in academic centers.)

Activated charcoal is ineffective or contraindicated in the following: (Think "CHEMICAL CamP")

- Caustics
- Hydrocarbons (and most water-soluble compounds)
- Electrolytes (common ones)
- Metals
- Iron
- Cyanide
- Alcohols (including ethanol)
- Lithium
- Camphor
- Phosphorus

Gastric lavage is not routine anymore. It can be considered when a life-threatening ingestion has occurred within 30–60 minutes and other treatment is unavailable. It is recommended only for persons who can tolerate a large-bore tube. One worrisome complication is pulmonary aspiration. Contraindications for gastric lavage include caustic (both acids and alkalis), hydrocarbon, and sharp-item ingestions. If no pill fragments are seen, this does not rule out toxic ingestion.

Whole bowel irrigation (WBI) is the flushing of the entire GI tract with polyethylene glycol until the effluent turns clear. Patients may experience GI discomfort. It's used, for example, in cases where activated charcoal is expected to be ineffective or extended release of the toxin is anticipated.

Cathartics decrease GI transit time, but clinical studies to date have not shown improved outcomes. Sorbitol is frequently given with activated charcoal, but cathartics are not used alone because they cause electrolyte imbalances.

Ipecac is an over-the-counter drug that induces vomiting. It is no longer recommended for use in homes or clinical settings.

## ACETAMINOPHEN INGESTION

Acetaminophen is the most commonly used analgesic in the U.S. and leads to 12% of pediatric poisoning deaths. It is rapidly absorbed and then metabolized in the liver to nontoxic metabolites using glutathione. With overdoses, glutathione stores are overwhelmed and toxic metabolites accumulate.

An acute toxic dose in a child < 12 years of age is about 150 mg/kg, and in adolescents and adults it is ~ 15 grams. Note: Chronic, repeated large doses may lead to toxicity as well.

The symptoms of acetaminophen overdose occur as follows:

- 0–24 hours: If any signs, nausea, vomiting; normal liver function tests (LFTs).
- 24–48 hours: Asymptomatic; maybe RUQ pain; LFTs may begin to increase.
- 48–96 hours: Peak of symptoms; AST > 20,000, prolonged PT, death from hepatic failure or coagulopathy.
- 4–14 days: Recovery or death; symptoms resolve in survivors.

When a child/adolescent presents with acetaminophen overdose, begin the ABCs first; and then, to prevent further absorption, give activated charcoal. Next, check acetaminophen levels 4 hours after ingestion and use the Rumack-Matthew nomogram (Figure 3-1) to determine if toxicity is likely. If toxicity is possible or probable using the nomogram, start IV N-acetylcysteine (NAC) or acetylcysteine (Mucomyst®) within 8 hours of ingestion. Continue a full course of treatment if started, and get a toxicology consult. The IV form is just as effective as the oral form and is better tolerated. Don't forget to consider co-ingestions. There is no need to follow acetaminophen levels after toxicity has been determined. However, you do need to follow AST, ALT, PT, and PTT levels.

## ANTICHOLINERGIC INGESTION

These agents include some antihistamines (e.g., diphenhydramine), antidepressants (e.g., amitriptyline, imipramine), antispasmodics, antiparkinson agents, atropine and its relatives (in OTC sleep meds), and toxic plants (mushrooms, jimson weed, deadly nightshade).

**Quick Quiz**

- Name some products that activated charcoal would not be useful in absorbing.
- Is ipecac recommended for home use?
- Know acetaminophen poisoning!!!!!
- What are the symptoms of an anticholinergic ingestion?
- Know iron poisoning!

The symptoms are key!:

| | |
|---|---|
| Dry as a bone: | ↓ sweating, ↓ urine output |
| Red as a beet : | Flushing |
| Blind as a bat: | Mydriasis |
| Mad as a hatter: | Agitation, seizures |
| Hot as a hare: | Hyperthermia |

Treat with activated charcoal. Use of physostigmine is controversial because side effects include seizures, bronchospasm, and decreases in blood pressure and heart rate.

## CARBAMAZEPINE INGESTION

Carbamazepine ingestion (though rare, it is in the content specifications for the Boards) results in CNS depression, and symptoms usually occur within 6–24 hours.

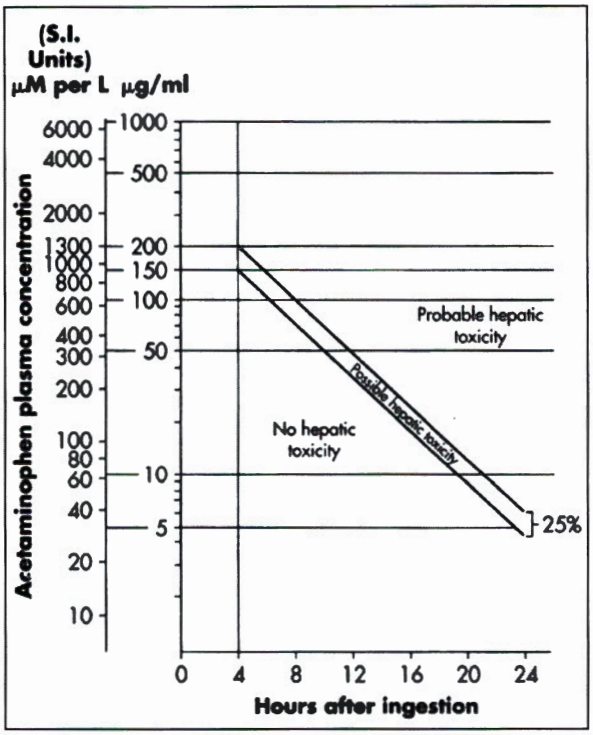



**Figure 3-1:** Rumack-Matthew Nomogram

Mild ingestions may just result in drowsiness, vomiting, ataxia, slurred speech, and/or nystagmus. Severe intoxications can result in coma, seizures, and respiratory depression. The optimal time for post-ingestion testing is 2–4 hours. Follow carbamazepine levels (there will be a delayed peak for 24–72 hours) and renal function. Treat with activated charcoal and supportive measures.

## CLONIDINE INGESTION

Clonidine is used to treat a wide array of problems; e.g., hypertension, ADHD, nicotine withdrawal symptoms. It is an α-2 adrenergic agonist. In children, even a dose as small as 1 pill can cause toxicity. Dermal patches contain a high dose. Symptoms occur within an hour of ingestion and mimic opioid toxicity: CNS and respiratory depression with pinpoint pupils. Remember the miosis mnemonic: COPS (Clonidine is in COPS). There may be bradycardia (even 1st or 2nd degree AV block) and hypotension—it is an antihypertensive—or, paradoxically, they may develop hypertension.

Serial ECGs and blood gases help determine the need for supportive care, such as intubation, atropine, fluid, and pressors. Activated charcoal may be appropriate and whole bowel irrigation may be indicated for children who ingest a patch. Naloxone has had mixed success—if it works, continuous infusion is indicated. Toxicity usually resolves within 24 hours.

## IBUPROFEN INGESTION

Ibuprofen inhibits prostaglandin synthesis and may result in GI irritation, reduced renal blood flow, and platelet dysfunction. A dose of < 100 mg/kg does not usually cause toxicity, while a dose of > 400 mg/kg can cause seizures and coma. Symptoms generally occur within 4 hours of ingestion and resolve in 24 hours. Nausea, vomiting, epigastric pain, drowsiness, lethargy, and ataxia can occur. Serious complications are rare but can include anion gap metabolic acidosis, renal failure, coma, and seizures. Monitor renal function and acid-base status. Use activated charcoal and supportive care.

## IRON INGESTION

Iron is a common pediatric poisoning. Most serious ingestions involve adult iron pills, especially prenatal vitamins. Iron is corrosive to gastric and intestinal mucosa. An elemental iron ingestion is classified by dose: 20 mg/kg is mild; 40 mg/kg is moderate; > 60 mg/kg is severe.

There are 4 overlapping phases of iron toxicity:

1) GI stage (30 mins to 6 hours)
   - Nausea, vomiting, diarrhea, abdominal pain
   - Hematemesis and bloody diarrhea in severe cases
   - Symptoms are due to the direct damage to the GI and intestinal mucosa

2) Stability (6–24 hours)
3) Systemic toxicity (12–24 hours)
   - Hypovolemic shock, cardiovascular collapse
   - Severe metabolic acidosis (positive anion gap)
   - Hepatic failure, jaundice
   - Coagulation disruption worsens GI bleeding
   - Coma
4) GI/pyloric scarring (2–6 weeks post-ingestion)

X-ray may show pill fragments, but liquid preparations and chewables are not generally visible. Obtain a serum iron level at 4–6 hours post-ingestion: A nontoxic ingestion is < 350 mcg/dL; a toxic ingestion is > 500 mcg/dL.

Treatment includes supportive care. Chelation with IV deferoxamine is recommended if the serum iron level is > 500 mcg/dL or if the patient has moderate or severe symptoms regardless of the iron level. You can consider whole bowel irrigation or endoscopic gastric removal. Do not use: gastric lavage (tablets are too large), ipecac, activated charcoal (does not bind iron), oral bicarbonate, magnesium hydroxide, or oral deferoxamine.

## OPIATE INGESTION

Common opiates include morphine, heroin, methadone, propoxyphene, codeine, and meperidine. Most cases present from drug abuse.

Note: The classic triad of coma, respiratory depression, and pinpoint pupils (miosis—recall COPS) suggests opioid poisoning.

Other expected findings:

- Analgesia (of course)
- Altered mood
- GI issues: decreased GI motility, nausea and vomiting, abdominal pain (from increased colonic and biliary tone), and even increased anal sphincter tone

Therapeutic doses of morphine have no direct effect on blood pressure and heart rate; but use may lead to histamine release, which dilates vessels, leading to orthostatic hypotension.

Treatment consists of ABCs, intubation if necessary, and naloxone as needed. Naloxone may precipitate withdrawal syndrome if the patient is physically dependent on the drug.

## PHENOTHIAZINE INGESTION

Phenothiazines include drugs used as tranquilizers and antiemetics: promethazine, prochlorperazine, and chlorpromazine. Dose-dependent effects may include anticholinergic effects, as well as disturbances of CNS (depression or seizures), temperature (up or down), and blood pressure (up or down). Moderate cases may present with "cogwheel rigidity" of neck, biceps, or quadriceps. Severe cases may include cardiac conduction abnormalities (especially long QT interval).

The notable dose-independent effect is dystonic reaction: spasms of the neck, tongue thrusting, and oculogyric crisis.

Treat with charcoal; support blood pressure as needed. Treat dystonia with diphenhydramine IV or IM.

## SALICYLATE INGESTION

Salicylates are not just in aspirin. Consider salicylate poisoning if the patient took an OTC cold medication, antidiarrheal, herbal preparation, or topical analgesic containing oil of wintergreen. Overdose may be acute or chronic. Ingestion of > 150 mg/kg puts the patient at risk. 3 chief systems are affected:

1) GI (nausea, vomiting)
2) Respiratory (hypernea leading to respiratory alkalosis)
3) If severe, CNS (agitation, confusion, and coma)

Ironically, moderate overdoses can cause fever. Tinnitus is the most specific finding. Death occurs from pulmonary or cerebral edema, electrolyte imbalances, and hypovolemia, leading to cardiovascular collapse.

Because salicylates are weak acids that uncouple oxidative phosphorylation, they cause a positive anion gap and metabolic acidosis. Check:

- Blood gas: The patient may have a respiratory alkalosis, metabolic acidosis, or a mixed picture.
- Electrolytes: Look for low glucose, potassium, volume.
- Salicylate level in serum:
  - > 30–50 mg/dL is potentially toxic—follow the patient.
  - > 50–100 mg/dL is usually symptomatic.
  - > 100 mg/dL indicates serious toxicity—CNS and respiratory.

Board tip: Ferric chloride with any salicylate turns urine purple or brown.

Management includes supportive care. Monitor levels every 2–3 hours. Follow blood gases, electrolytes, and coagulation studies, as well as serial ECGs. The Done nomogram is not used.

Use activated charcoal, but be aware that salicylate tablets can form a bezoar that may even require surgical removal. Rehydrate and replace electrolytes. To enhance elimination, alkalinize the urine; i.e., give bicarbonate to raise urine pH > 8. Hemodialysis may be helpful in serious cases.

## THEOPHYLLINE INGESTION

Theophylline is used less and less, but still commonly appears on the Boards. Remember that the therapeutic

**Quick Quiz**

- What is the classic triad of opiate ingestion?
- What drug is useful in treating dystonic reactions in phenothiazine ingestion?
- What are the acute findings of salicylate ingestion?
- Why is bicarbonate given during therapy for treatment of salicylate ingestion?
- Name the common electrolyte abnormalities found in theophylline toxicity.
- Name the cardiac findings associated with tricyclic antidepressant ingestion?
- What agent is used in treating tricyclic antidepressant overdose that will help prevent dysrhythmias?
- What lab test is useful in diagnosing carbon monoxide poisoning?

level for theophylline is in a small window, and upper normal levels can cause symptoms of toxicity. Normal therapeutic levels are usually 10–20 mcg/dL. With 20–50 mcg/dL, nausea, vomiting, and muscle tremor can occur. > 60–70 mcg/dL can result in seizures and arrhythmias.

Electrolyte abnormalities are common, including:

- ↑ glucose and calcium
- ↓ potassium and phosphate
- metabolic acidosis

Management centers on the ABCs and carefully correcting the electrolyte abnormalities. Repeated doses of activated charcoal are effective. Hemodialysis is effective in serious cases.

## TRICYCLIC ANTIDEPRESSANT INGESTION

Tricyclic antidepressants (TCAs) have anticholinergic activity. They inhibit cardiac fast sodium channels. Symptoms occur within 30 minutes to 6 hours. Ingestion of 10–20 mg/kg is moderate to serious.

In children, CNS effects are more prominent and include drowsiness, lethargy, seizures, and coma. Cardiac effects are:

- Tachycardia, including ventricular tachycardia
- Hypertension or hypotension
- Widened QRS
- Prolonged QT

Be aware of CCCA in tricyclic antidepressants:

- Coma
- Convulsions
- Cardiac dysrhythmias
- Acidosis

For treatment, it is important not to induce emesis because of the increased risk of aspiration. Give activated charcoal and add sodium bicarbonate to alkalize the serum to 7.45–7.5 to prevent dysrhythmias. Most dysrhythmias that occur with TCA overdose will respond to lidocaine. ECG monitoring is important. Physostigmine is not given—it worsens ventricular conduction and lowers seizure threshold. On the Boards, be suspicious of a younger sibling who "got into" the medications of a depressed relative.

# ENVIRONMENTAL INGESTIONS

## OVERVIEW

Environmental ingestions include the non-pharmaceuticals and will be discussed in further detail with each type of ingestion.

## CARBON MONOXIDE

Carbon monoxide (CO) binds to hemoglobin preferentially and displaces oxygen. It also impairs oxygen release (shifts the curve to the left) and impedes oxygen utilization. CO is colorless and odorless; poisoning occurs in settings of improperly vented stoves and auto exhaust.

Patients present with headache, malaise, nausea, and may appear to have "the flu." On the Board exam, look out for the whole family with these types of symptoms and for patients with "cherry red" skin.

Measure carboxyhemoglobin levels by cooximetry on an arterial blood gas sample. Most patients with carboxyhemoglobin levels > 15–20% have symptoms. Note: Pulse oximetry and $P_aO_2$ may be falsely normal!

Symptoms at higher levels include:

- Moderate exposure (20–40%): severe headache, dizziness, visual changes, syncope, vomiting, and ataxia
- Severe exposure (above 60%): coma, seizures, and death

Treatment is with high-flow mask oxygen (well-fitting nonrebreather with a reservoir). The CO half-life is about 5 hours in room air, but 60–90 minutes on 100% oxygen. Correct metabolic acidosis and underlying anemia. Severely affected patients may benefit from hyperbaric oxygen. Also consider cyanide poisoning in patients from a house fire, especially in those patients with persistent metabolic acidosis.

## CAUSTIC SUBSTANCE INGESTION

Generally, these fall into either alkaline or acidic agents.

Alkaline agents and characteristics:

- Bleach, ammonia, cleaners for ovens and drains, automatic dishwasher detergent, hair relaxers, lye (everyday household bleach [5%] is only an irritant)
- Tasteless
- Cause severe, deep, liquefaction necrosis
- May lead to scar tissue with strictures

Acidic agents and characteristics:

- Toilet bowl cleaner, grout cleaner, rust remover, automotive battery liquids, metal cleaners (gun bluing—note: This has been an ABP content specification!)
- Bitter taste
- Coagulation necrosis (superficial)
- May lead to thick eschar formation, severe gastritis, metabolic acidosis, or acute renal failure

Caustic ingestions cause major problems with esophageal and gastric inflammation and can potentially cause perforation. Note: Acids can cause severe gastritis, perforation, or late stricture even without severe mouth/esophageal burns. Stricture formation probably occurs in 20% of caustic ingestions.

Symptoms with both alkaline and acidic ingestions include:

- GI tract irritation: vomiting, drooling, refusal to drink, oral burns, dysphagia
- Respiratory tract irritation: stridor
- Chest or abdominal pain

The absence of symptoms usually implies little or no injury; however, remember that absence of oral lesions does not preclude severe esophageal or stomach injury. 20–40% have no burns in the mouth!

Upper endoscopy is recommended in all symptomatic children and in all with visible burns in their mouth.

Treatment includes diluting with water or milk (Nelson's says this, but most Peds emergency medicine docs consider this fraught with hazard). Remove contaminated clothing and rinse the affected skin. Check chest and abdominal film for pneumomediastinum and aspiration pneumonitis. Start an IV if needed for fluids and analgesia. Endoscopy should be done within 24–48 hours. After endoscopy, observe for complications. Steroids are usually not indicated. Strictures require treatment with dilatation and possible surgical resection.

Do not neutralize, induce emesis, do gastric lavage, or give activated charcoal. More in the Gastroenterology & Nutrition section.

## HYDROCARBON INGESTION

Hydrocarbons are low-viscosity compounds; ingestion can lead to pulmonary aspiration. They are divided by risk (common examples):

- Nontoxic unless gross aspiration: mineral, baby, or suntan oils
- Aspiration hazards: mineral spirits, lamp oil, gasoline, kerosene, furniture polish, lighter fluid, turpentine
- Systemic toxins: benzene, toluenes, and hydrocarbons that serve as vehicles for pesticides

Clinical findings include coughing, choking, gagging, wheezing, and severe respiratory distress, as well as mild CNS depression and fever. These patients may have a high WBC. Recall: CXR may be normal for 24 hours after exposure!

Don't forget dermal decontamination! Generally, for these exposures in the asymptomatic child, you will observe for 6–8 hours. These children can be discharged if they are still asymptomatic, the CXR is normal, and they have a normal oxygen saturation. If symptomatic or the CXR is positive, start supportive care with airway control and the other ABCs. ARDS is likely in severe cases. Do not give ipecac, gastric lavage (except in very special circumstances), activated charcoal, steroids, prophylactic antibiotics, or epinephrine (will induce ventricular fibrillation).

## ETHANOL INGESTION

Ethanol is found in many everyday items in the household besides spirits and liquor, including mouthwash and perfume. Signs and symptoms of ethanol ingestion include:

- CNS disturbances: depression (slurred speech, ataxia, and stupor to coma), seizures
- Respiratory depression
- GI disturbances: nausea, vomiting
- Hypothermia
- Hypoglycemia (inhibits hepatic gluconeogenesis, leading to hypoglycemia in children < 5 years)

A high osmolal gap should make one suspicious for ingestion of ethanol (or methanol, ethylene glycol, or isopropyl alcohol). Diagnosis is confirmed by ethanol level.

Treatment includes the usual ABCs and giving intravenous fluids. Treat hypoglycemia or hypokalemia as needed. Activated charcoal is not recommended for ethanol ingestion alone, but may be considered if a co-ingestion is suspected. Hemodialysis can be done but is rarely needed. Consider screening for other toxins in the face of presumed isolated ethanol ingestion!

**Quick Quiz**

- True or false? Acid ingestions can cause severe gastritis, perforation, or late stricture even without severe mouth/esophageal burns.
- Know the similarities and differences between ethanol, methanol, and ethylene glycol ingestion!
- What are the symptoms of organophosphate ingestion? Treatment?

## METHANOL INGESTION

Methanol is found in windshield washer fluid, deicer, antifreeze, canned heat, picnic stove fuel, and as a fuel additive. If ingested, it is metabolized to formic acid, which inhibits mitochondrial respiration via alcohol dehydrogenase.

Symptoms:

- Initial nonspecific complaints (malaise, headache, abdominal discomfort, nausea, and vomiting)
- 24 hours later, the child will develop:
  - Visual disturbances with blurry vision and photophobia (described as a snowstorm)
  - Optic nerve damage leading to blindness
  - CNS depression
  - Severe metabolic acidosis (high anion gap)

Look for a triad of:

1) Visual complaints
2) Abdominal pain
3) Metabolic acidosis (without lactic acidosis or ketonuria)

Because of rapid absorption, gastric decontamination and activated charcoal generally do not work. Treat the metabolic acidosis with sodium bicarbonate. Competitively inhibit alcohol dehydrogenase with IV fomepizole (fewer side effects, easier dosing, but more costly than ethanol, but generally has supplanted ethanol as the drug of choice). Folate enhances the metabolism of formic acid and so it is an adjunctive therapy. Hemodialysis (for methanol > 25 mg/dL or severe symptoms) is effective in removing the agent and will help correct the metabolic acidosis.

## ETHYLENE GLYCOL INGESTION

Ethylene glycol is sweet and found in antifreeze, radiator fluid, and other coolants. If ingested, it is metabolized to glycolic and oxalic acid—both toxic—via alcohol dehydrogenase. Oxalic acid chelates calcium; calcium oxalate crystals will be found in the urine. Urine may fluoresce with a Woods lamp.

There are 3 stages of intoxication:

- Stage 1: CNS manifestations—appears drunk with vomiting, drowsiness, slurred speech, lethargy; metabolic acidosis
- Stage 2: coma, cardiac and respiratory problems—tachypnea, cyanosis, pulmonary edema, ARDS; death can occur
- Stage 3 (after 24–72 hours): renal failure

Like methanol, ethylene glycol ingestion leads to metabolic acidosis (without lactic acidosis or ketonuria) and high osmolal gap.

Because of rapid absorption, gastric decontamination and activated charcoal generally do not work.

Treatment includes 4 foci:

1) Treat the metabolic acidosis with sodium bicarbonate.
2) Competitively inhibit alcohol dehydrogenase with IV fomepizole (preferred; if not available, then IV ethanol).
3) Thiamine and pyridoxine shunt metabolism to less toxic metabolites; therefore they are an adjunctive therapy.
4) Hypocalcemia is common and may also require therapy.

Hemodialysis is needed for ethylene glycol > 25 mg/dL or severe symptoms.

## ORGANOPHOSPHATE INGESTION

Organophosphates are found in pesticides (e.g., diazinon, malathion) and nerve agents (e.g., sarin). They inhibit cholinesterase enzymes and can form permanent bonds over 1–3 days. It can take weeks to months for enzyme regeneration.

Inhibition of cholinesterase leads to the cholinergic toxidrome (DUMBELS):

- Diarrhea
- Urination
- Miosis (pinpoint)
- Bronchorrhea/Bronchospasm
- Emesis
- Lacrimation
- Salivation
- (Basically, miosis + an outpouring of every bodily fluid)

Other symptoms can develop, including "nicotinic effects": twitching, weakness, and respiratory weakness. Confusion, coma, convulsions, and slurred speech can occur as well.

Diagnosis is confirmed by decreased RBC cholinesterase activity (but don't wait to treat).

For treatment, it is important for health care workers involved to wear protective clothing (surgical mask and latex gloves are not effective). Decontaminate the child's clothing and skin with soap and water. Activated charcoal is recommended. Begin ABCs and give 2 antidotes:

1) Atropine: may need large doses; repeat until effective.
2) Pralidoxime (2-PAM): hydrolyzes the bond if given before it becomes permanent. 2-PAM doesn't cross blood-brain barrier, so always use with atropine.

Diazepam and other benzodiazepines can be used for CNS symptoms. Symptoms can persist for weeks without treatment.

## PLANT EXPOSURES

For the Board exam, you need to know about a few toxic plants:

- Digitalis effects: foxglove, lily of the valley, and oleander
- Atropine effects: jimson weed, deadly nightshade
- Cyanide-like: pear and apple seeds, peach pit, bitter almond
- Liver toxicity: mushrooms (lethal ones will provoke symptoms > 6 hours after ingestion)
- Oral pain: dieffenbachia and philodendron
- Mild GI symptoms: poinsettia, mistletoe, and holly

# BITES

## CAT / DOG BITES

More than 4.5 million people in the U.S. are bitten by dogs each year, only about 50% of which are reported. Surveys indicate that 55% of boys and 39% of girls between the ages of 4 and 18 report being bitten during their lifetimes. Dog bites of children are most frequently (75%) on the head or neck. Risk factors for infection include location (hand), type (puncture), interval to care (> 24 hrs), and animal (cat).

Cat-bite infections are generally due to *Pasteurella* and *Staphylococcus aureus*; dog bites carry the same organisms plus DF-2. The most common organism in cat-bite wounds is *Pasteurella multocida*, which is found in 60–75% of "normal" cats' mouths.

Puncture wounds are deceptive and are often more extensive than recognized on initial examination. Radiographs may be necessary when deep puncture wounds are close to bone or joints. These initial x-rays are also useful as comparison films in case complications occur down the road and osteomyelitis or septic arthritis appears to be developing.

All wounds require thorough cleaning with soap and water with copious saline irrigation. Rabies immunization should be considered based on type of wound, animal involved, and local health department data (see Rabies in the Infectious Disease section). Tetanus immunization also should be considered if indicated (again, see the Infectious Disease section).

Prophylactic antibiotic use is debated. It may be justified for certain body areas (hand, foot, bone, joint, tendon), biters (humans, cats), patients (immunocompromised), or wounds sutured closed. Because cat bites are usually puncture wounds and have a tendency to go "deep," most should be treated with amoxicillin-clavulanate or, if the child is penicillin-allergic, clindamycin plus trimethoprim/sulfamethoxazole (or ciprofloxacin if > 18 years of age). Dog bites sometimes can be observed without treating with antibiotics—because they are more commonly "crush" injuries rather than deep piercing injuries; however, if a dog-bite wound already looks infected, then the same antibiotics are recommended. Some recommend giving the first dose of antibiotic intravenously, using ampicillin-sulbactam or a carbapenem (imipenem, meropenem, or ertapenem).

Primary wound closure is generally not recommended. Most experts recommend high-risk wounds (e.g., not cleaned for 12 hours, need extensive debridement, or for which prophylactic antibiotics are discussed above) should not be closed.

Referral to a surgeon or specialist should be considered if any of the following are noted:

- Hand bites, except those that are superficial and fresh
- Extensive infection at the bite site
- Damage to tendon, cartilage, bone, or joint capsule
- Wounds that might cause disfigurement or require plastic surgery
- Young children with head injuries from bites due to large dogs (the severity of the injury may be underestimated)

## SNAKE BITES

More than 95% of snake bites in the U.S. are from pit vipers (e.g., rattlesnakes, water moccasins, copperheads). They have a triangular head, elliptical eyes, and a pit between each eye and nostril. Venom from these snakes causes tissue necrosis, vascular leak, coagulopathies, and neurotoxicity. Most bites are sustained by young adult males while handling a snake, but children are at risk for serious sequelae due to their smaller body mass.

Signs and symptoms will generally develop within 2–6 hours with severe pain, nausea, vomiting, weakness, muscle fasciculations, and coagulation abnormalities.

Treatment relies on identification of the snake (or the most likely, based on local epidemiology). Initial therapy is to immobilize the body part, keep the wound below the heart if possible, and provide wound pressure. Do not apply ice or a tourniquet of any kind nor use excision and suction (John Wayne may have done it but it is not recommended today). Place an IV for fluids and check coagulation studies and CBC with RBC

**Quick Quiz**

- What is a potential effect of eating foxglove?
- Name the 2 most common organisms found in a cat-bite wound infection.
- Explain the differences between first-, second-, and third-degree burns.
- Be able to determine burn surface area in an adolescent and a child.

morphology. Give pain medication and update the tetanus immunization, if indicated.

Pit viper antivenin (CroFab®) is indicated for minimal or moderate North American pit viper envenomation, preferably within 6 hours, for initial control of all venom effects. These include local (progression of swelling), systemic (e.g., vital sign or GI disturbance, oral paresthesia, or unusual tastes), and coagulation abnormalities. Although the clinician should be prepared to manage anaphylaxis, skin testing and pretreatment are not routinely recommended before use of the antivenin (unlike horse serum-based antivenom).

## SPIDER BITES

Brown recluse spiders, which are responsible for most U.S. spider bites, are usually seen in the Southeast and Southwest; hide in woodpiles, attics, and closets; and have a leg span of 25 mm. Adults have a dark violin pattern on the dorsal front portion of the body (cephalothorax); thus, they are called "fiddleback" or violin spiders. Their venom lyses cell walls locally. Initially, the bite is painless; pain develops at the site 2–8 hours later. A hemorrhagic blister develops and progresses into a large ulcer. Think of the "u" in recluse for ulcer! Patients rarely have systemic symptoms, e.g., fever, chills, nausea, or vomiting. Treatment is with hydration and local wound care. Surgical repair may be needed when necrosis is no longer spreading, which may take 8 weeks. Other treatments (e.g., dapsone, steroids) are not evidence-based.

Black widow spiders are found throughout the U.S. (except Alaska), hide in dimly lit, warm, dry outhouses and sheds, and are large, with a leg span of 40 mm. A mature female is black with a red/orange hourglass marking on the ventral surface. The black widow spider's venom is a neurotoxin. Initial signs and symptoms include pain at the site, muscle cramping, chest tightness, vomiting, malaise, sweating, abdominal pain (can mimic appendicitis), agitation, and hypertension. Treatment is with opiates, benzodiazepines, and—in severe cases—antivenin is available. Usually symptoms resolve in 24–48 hours. IV calcium is not effective.

# BURNS

## OVERVIEW

Burns are a leading cause of unintentional pediatric death. 18% of burns are due to abuse. Burn first aid includes ABCs, removal of clothing, washing off chemicals, and covering the burn with a clean dry sheet. For small burns, cold wet compresses may be applied; for large burns, do not use cold/wet compresses because this will lead to hypothermia. No grease or butter!

Burn depth is classified as:

- First-degree (superficial): red, blanches with pressure, dry, minor swelling and pain. They usually resolve in 5–7 days.
- Second-degree (partial-thickness): red, wet, very painful; often with blisters or blebs. The tissue underneath is still well perfused. It may take 2–5 weeks for these to heal.
- Third-degree (full thickness): dry, leathery, waxy, and have no pain associated with them. They require grafting in large areas or healing from the edges in smaller burn areas.

Measurement of burn surface areas follows the Rule of Nines (> 14 years old):

| | |
|---|---|
| • Head and neck: | 9% |
| • Each upper limb: | 9% |
| • Thorax and abdomen, front: | 18% |
| • Thorax and abdomen, back: | 18% |
| • Perineum: | 1% |
| • Each lower limb: | 18% |

Rule of Palm (< 10 years of age):

- Child's palm not including fingers = 0.5–1% body surface area
- Useful in smaller burns

## MINOR BURN CARE

First-degree burns require no therapy.

Partial-thickness (second-degree) burns:

- Clean with soap and water daily.
- Leave blisters intact if < 2 cm; debride when ruptured.
- Apply temporary skin substitutes if available.
- Antibiotic ointment (silver sulfadiazine [Silvadene®] or bacitracin)—not needed if transferring to trauma center.
- Facial burns may be left open.
- Pain control.
- Tdap if needed.
- Change dressing once daily.
- Reevaluate every 2–3 days because the burn may progress.

## MAJOR BURN CARE (THIRD-DEGREE)

ABCs and don't forget to also consider carbon monoxide.

IVF (ringers lactate usually) for burns > 15% body surface area.

Parkland formula:

- Ringers lactate: 4 mL/kg/percent of total burn surface area is given in the first 24 hours, with 50% of the volume in the first 8 hours and the rest over the next 16 hours.
- Add maintenance rate to this volume for smaller children.

For children < 20 kg, many recommend adding 5% dextrose to the ringers lactate to prevent hypoglycemia.

The chief reasons to refer a child to a burn center include:

- Extent: 2nd degree of > 30% total body surface area (TBSA); 3rd degree of > 10–20%.
- Burn site: 2nd or 3rd degree burns of hands, feet, face, perineum, major joints (anywhere that is important to cosmesis or function).
- Burn type: electrical, chemical, inhalation.
- Patient: with certain preexisting disorders, concomitant trauma (if the burn poses the major risk), or social or emotional needs (e.g., child abuse, substance abuse).
- The referring facility does not have qualified personnel or equipment.

## ELECTRICAL BURNS

Minor electrical burns are usually asymptomatic and require no evaluation. Use minor cleansing and antibiotic cream.

If there are burns to the oral commissure from an electrical cord bite, refer to a burn surgeon because of potential for labial artery bleeding 1 to 3 weeks after the injury.

High-voltage electrical burns may cause deep tissue injury, which can lead to myoglobin release and renal failure. Check for myoglobin in urine, serum creatine kinase, and an ECG. Refer to a children's burn center.

# SUBMERSION INJURY

Drowning ranks second to motor vehicles as a cause of accidental pediatric death. 40% occur in those < 5 years of age. Among 5–19-year-olds, drowning is more common in males and Caucasians.

With the initial swallowing of water, laryngospasm occurs. Loss of consciousness follows, with vomiting and aspiration in 90%. Hypoxia occurs and results in loss of circulation in 3–4 minutes, with CNS injury in 3–5 minutes. CNS injury is the most frequent cause of death. Pulmonary aspiration is usually minimal, but ARDS or pulmonary edema may develop. Hyponatremia is uncommon.

Hypothermia can be protective. Good outcomes have occurred in those with prolonged submersion in icy water (< 5° C) and who had a core body temperature of < 30° C. Cold water above this temperature is not protective.

Freshwater and saltwater drownings are managed the same way. Treatment includes the ABCs. Protect the spine if appropriate (abuse, diving injury). Vomiting is common, so use cricoid pressure or a nasogastric tube. Give oxygen, check a CXR, address metabolic acidosis. After cold water submersion, use warmed IVF and gastric and bladder lavage. For freezing-water drowning, continue resuscitative efforts until the core temperature is 32° C ("They're not dead 'til warm and dead").

Monitor all near-drowning patients for 6–24 hours; delayed respiratory symptoms will manifest by then.

# HEAD INJURY

## OVERVIEW

Head injuries are common. For all children, falls are the most common cause of head injury, although motor vehicle trauma is the most common cause of serious head injury. Symptoms to watch for include: vomiting, lethargy, headache, irritability, and behavioral changes.

Physical findings indicating more serious injury include:

1) Scalp swelling or step-off
2) Basilar skull fracture (often associated with CSF leak, cranial nerve damage):
   - Raccoon eyes (Image 3-1)
   - Battle sign (Image 3-2)
   - Hemotympanum
3) Temporal bone fracture:
   - Bleeding from the external auditory canal
   - CSF otorrhea
   - Hearing loss
   - Facial paralysis
4) Pupillary changes; papilledema doesn't develop immediately
5) Retinal hemorrhages (abuse!) or other bruises

There do not have to be significant signs of external head trauma to have a significant brain injury!

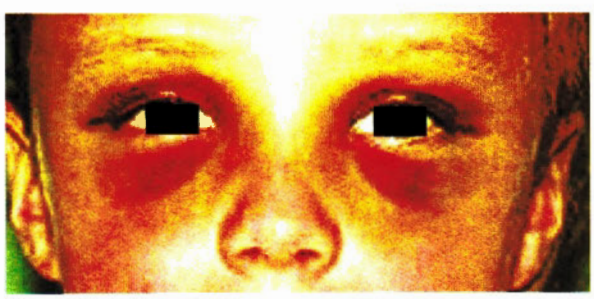


*Image 3-1: Raccoon Eyes*

**QuickQuiz**

- How do you manage a near-drowning victim?
- Describe findings in a basilar skull fracture.
- Describe findings in a temporal bone fracture.
- What do retinal hemorrhages indicate?
- In a head injury case, when should you definitely order a CT scan of the head?
- In a head injury case, when is it okay to send the patient home for observation?

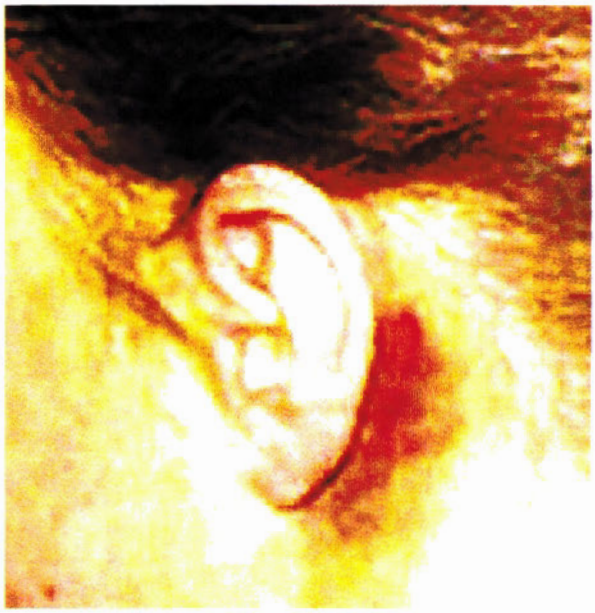
## IMAGING OF THE HEAD

The guidelines for evaluating children after head trauma are evolving. CT is the preferred study when intracranial injury (ICI) is suspected. Plain film doesn't assess for ICI; MRI availability is limited and they are hard to get quickly.

Most children with minor head trauma and without symptoms do not need imaging. For example, CT is not necessary for an older child with a scalp hematoma unless there are other findings suggestive of skull fracture (e.g., step-off).

When to definitely order a CT scan (high risk):

- History of
  - Loss of consciousness > 1 minute
  - Seizure after event
  - Persistent/progressive vomiting
  - Underlying condition predisposing to ICI
- Exam reveals
  - Depressed mental status
  - Focal neurological signs
  - Signs of skull fracture
  - Irritability
  - Bulging fontanelle
- Suspicion of abuse



Courtesy of Thomas Ky, marziek, MD

*Image 3-2: Battle Sign*

Some examples of signs of skull fracture include: Battle sign (mastoid ecchymosis), hemotympanum, drainage of CSF as rhinorrhea, "raccoon's eyes" (periorbital ecchymosis), or cranial nerve palsies.

Intermediate-risk patients may be managed with head CT or observation with reevaluation. These patients have the following:

- Loss of consciousness < 1 minute
- Vomiting 3–4 times
- Lethargy or irritability now resolved
- Behavioral changes
- Mechanism: higher force, fall onto a hard surface, or unknown, unwitnessed, vague
- Hematoma, especially large or non-frontal
- Nonacute skull fracture

When to discharge with instructions (low risk):

- Mechanism of injury is low energy (e.g., fall of < 3 ft)
- No signs or symptoms
- No loss of consciousness

No patients involved in a motor vehicle collision or with unwitnessed head trauma are placed in this category!

Note: Only high- and low-risk groups are highlighted; others are controversial and less likely to appear on Board exams.

## CONCUSSIONS

Concussion injury questions on the Boards usually are sports-related. In the "game" situation, ask the player about the game (e.g., what period is it, the score, the team they are playing) instead of asking for name and phone number. A concussion is defined as a traumatic alteration in mental status and can result in any or all of these: disturbance of vision, loss of equilibrium, amnesia, headache, cognitive dysfunction, nausea, vomiting, or transient loss of consciousness. Concussion grading systems vary. A good version is offered in Table 3-1. For Grade I, there are symptoms for less than 15 minutes; for Grade II, there is no loss of consciousness, and symptoms present longer than 15 minutes; and for Grade III, there is loss of consciousness.

**Table 3-1:** Concussion Grades

| Grade | Confusion | Amnesia | Loss of Consciousness |
|---|---|---|---|
| I | Yes | No | No |
| II | Yes | Yes | No |
| III | Yes | Yes | Yes |

A major concern is prevention of subsequent concussions. "Second-impact syndrome" occurs when 2 head injuries occur within a day. This can result in loss of auto-regulation of cerebral blood flow, with rapid development of increased intracranial pressure and, ultimately, death. More commonly, repeat concussions before complete recovery lead to an accrual of neuropsychologic defects. So, a big question is, "When can the kid return to sports?" (Table 3-2)

**Table 3-2:** Concussion and Time Before Return to Contact Sports

| Grade | Minimum Time to Return To Play | Time Asymptomatic |
|---|---|---|
| I | 20 minutes | At the initial Exam |
| II | 1 week | 1 week |
| III | 1 month | 1 week |

There are several sets of recommendations regarding post-concussion "return to contact sports"; e.g., the American Academy of Neurology (1997), Colorado Medical Society (1991), and Cantu (2001). Guidelines agree that athletes suspected of having a concussion should be removed from sports participation immediately, and athletes should not return to play while signs or symptoms of concussion are present. Also, athletes who have any loss of consciousness, display any symptoms of concussion lasting more than 15 minutes, or who have posttraumatic amnesia should not be permitted to resume sports participation until asymptomatic for at least one week. An emergency department evaluation is indicated for any athlete who suffers loss of consciousness.

The guidelines differ regarding management of return to sports after subsequent concussions. As examples, the Cantu guidelines recommend terminating play for the season after a third concussion of any grade, while the AAN allows for return to play after a one-to-four week interval (depending on the grade of the concussion).

## LACERATIONS

Irrigation is the best way to clean a laceration. Make sure that tetanus immunization is up to date. On the scalp, do not use topical skin adhesives (e.g., Dermabond®). Do not use LET (lidocaine, epinephrine, tetracaine) gel on fingers, toes, nose, ears, or penis. Do not shave eyebrows. Eyelid lacerations require an ophthalmologist for tear duct repair. Lining up the vermillion border requires experience. A clenched fist hand laceration is at high risk of infection and should be referred to a hand surgeon.

Potential complications of lacerations include:

- Tendon laceration with loss of function
- Arterial or vascular compromise
- Infection
- Limited flexibility
- Cosmetic considerations; e.g., keloids, scarring

## FRACTURES

### OVERVIEW

Fractures are common in pediatrics. Growth plate injuries (15% of all childhood fractures) follow the Salter-Harris classification (Figure 3-2).

Types I (epiphysis slips or separates from the metaphysis) and II (a bit of metaphyseal bone separates with the epiphysis) are common and have excellent prognosis because, while they extend through the growth plate, the germinal layer is usually left intact—so growth disturbance is uncommon. Undisplaced fractures simply require immobilization in a cast.

Types III (a fracture through the epiphysis extending to the epiphyseal plate) and IV (a fracture that extends from the articular surface, through the epiphysis, and through the metaphysis) are uncommon. A precise reduction is required with an orthopedic consult. Involvement of

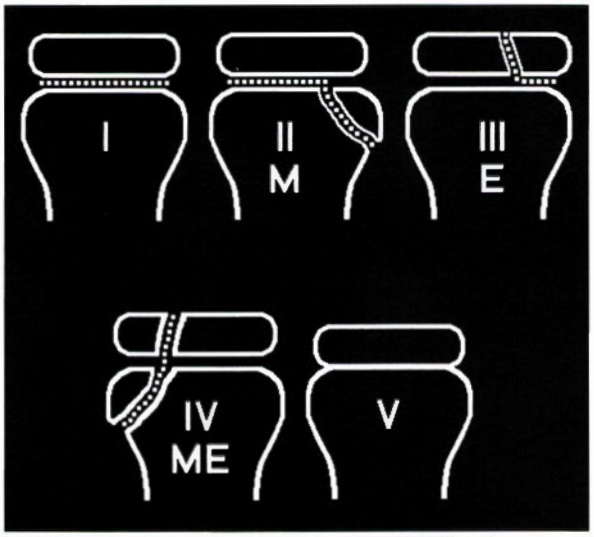



**Figure 3-2:** Salter-Harris Classification of Fractures

**QuickQuiz**

- Explain the Salter-Harris classification of fractures.
- What is the treatment for a greenstick fracture?
- Where is the most common place for a "buckle" fracture to occur?
- In a 7-month-old infant with a spiral fracture of the left femur, what should you suspect?
- How do you treat a clavicle fracture?

the articular surface and germinal layer makes growth disturbance and functional impairment more likely if not managed correctly. This manipulation will usually involve an anesthetic.

Type V is a crush injury to the epiphysis. It is rare and has a poor prognosis due to disruption of the blood supply to the epiphysis. Unfortunately, this is usually diagnosed retrospectively when limb deformity becomes apparent.

## GREENSTICK FRACTURE

This incomplete fracture is very common in children. The cortex is intact on one side. Anatomic reduction may require this cortex to be disrupted. If there is no deformation, immobilization alone may be effective therapy (Image 3-3).


*Image 3-3: Greenstick Fracture*

## TORUS (BUCKLE) FRACTURE

This is a common fracture in children. Compression of the bone produces a "torus" or "buckle" of the metaphysis. It is most common in the distal radial metaphysis. It usually heals with 3 weeks of immobilization (Image 3-4).

## SPIRAL FRACTURE

This sounds just like its name, with a fracture having a curvilinear course. A spiral fracture of the tibia is called a "toddler's fracture" because it is most common in toddlers. Think abuse in a child who is not walking yet, especially if it is the femur (Image 3-5)!

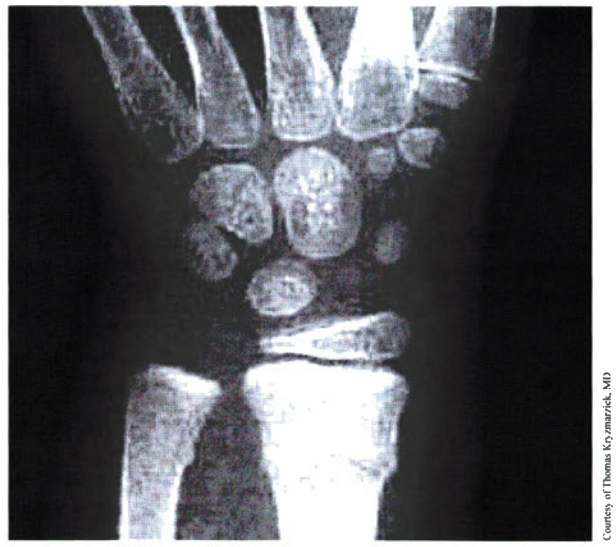



*Image 3-4: Buckle Fracture*

## CLAVICLE FRACTURE

This is the most common bone for children to fracture, but clavicle fractures are uncommon in those < 2 years of age (except newborns) and, thus, suspicious for abuse. Usually, it affects the middle and lateral portion of the clavicle. Most cases occur as a result of falling on an outstretched arm or direct trauma. Neurovascular injury is uncommon. Diagnosis is usually made by physical exam and/or radiograph (Image 3-6).

Treatment is immobilization of the affected arm for 2–3 weeks in a young child or a simple sling for 3–4 weeks in an older child. Most of the healing is spontaneous.

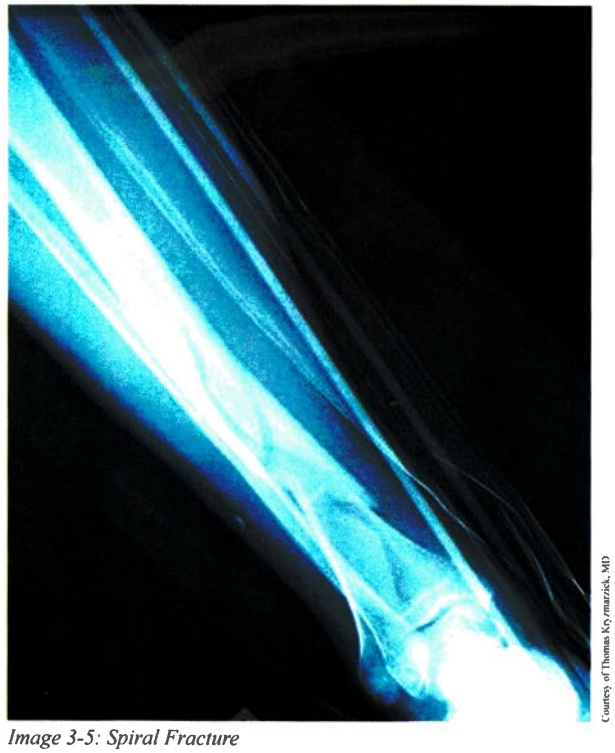



*Image 3-5: Spiral Fracture*

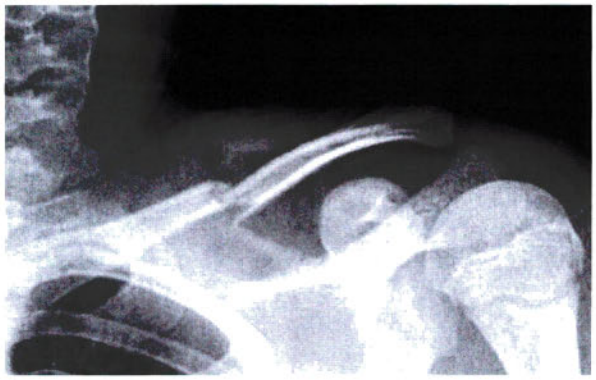



*Image 3-6: Clavicle Fracture*

## DISTAL HUMERUS (ELBOW) FRACTURE

The most common pediatric elbow fracture is the supracondylar fracture, which usually occurs after a fall on an outstretched hand or elbow. Look for the posterior fat pad sign. These fractures have a high risk of complication. If the fracture is displaced, evaluate for damage to the brachial artery, median nerve, or radial nerve. Neurovascular status must be monitored carefully! Refer to orthopedics (Image 3-7).

## FRACTURE COMPLICATIONS

We already mentioned that you should be on the lookout for neurovascular compromise with the supracondylar fracture.

Compartment syndrome, another concern, is most common with tibial and supracondylar fractures. The fracture (or other injury) results in hemorrhage or swelling in an enclosed fascial compartment. Then vascular injury results, leading to ischemia. Pulses may be normal. The clue is pain out of proportion to the fracture, especially pain remote to the fracture. Compartment syndrome requires an emergent orthopedic consult!

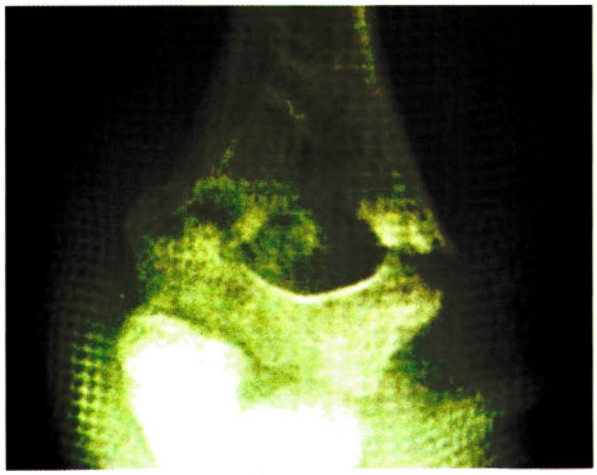



*Image 3-7: Distal Humerus Fracture*

Occult fractures are those not evident on x-rays. Possible clues:

- Clinical exam is consistent with a fracture, but no fracture is seen on x-ray.
- Persistent pain.
- Gait disturbances, especially in younger children!
- Treat as if fractured and follow x-rays.

## SUBLUXATION OF THE RADIAL HEAD

This is also known as "nursemaid's elbow." Usually, it occurs in children between the ages of 6 months and 5 years. The typical mechanism is axial traction on an extended and pronated arm—the annular ligament slides over the radial head. The child then holds the arm limply to the side. X-rays are not necessary. To fix the problem, supinate the forearm and flex the elbow, or, alternatively, you can pronate the forearm. Sometimes you hear a "click." After reduction, the child will return to normal functioning of the arm within 15 minutes.

## SHOULDER INJURIES

Acromioclavicular (AC) separation occurs in adolescents and most commonly occurs in a direct blow to the shoulder. There will be tenderness over the acromioclavicular joint. A sling is effective for minor injuries; refer to orthopedics if the injury is more severe. AC separation is rare in prepubertal children—they more commonly fracture the distal clavicle.

Shoulder dislocation, also rare in children < 12 years old, occurs commonly in adolescents. This results when the abducted, externally rotated shoulder is pushed posterior during contact sports. Swelling and deformity will occur over the shoulder anteriorly. Traction and countertraction can replace the shoulder; use muscle relaxants. Immobilize with a sling and swathe for several weeks. Involve orthopedics because of the high rate of recurrence.

## SPRAINS

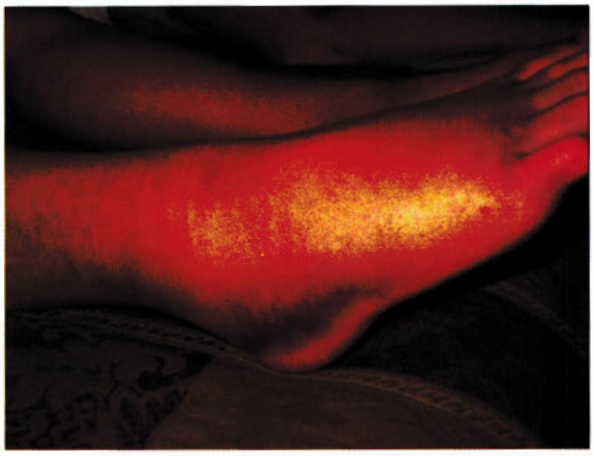
A sprain is an injury to the ligament around a joint when it is forced to move in an unnatural position. Sprains are rare in prepubescent children because the ligament is stronger than the growth plate; thus, these children more often get avulsion fractures rather than sprains. The most common sprains are of the ankle and fingers. On physical examination, the key thing to look for is tenderness (Image 3-8). Swelling and bruising are common as well. Ankle sprains are graded on findings in Table 3-3.

Although Ottawa ankle rules can be applied, in general obtain x-rays of the ankle to rule out a fracture. Treat with RICE (Rest, Ice, Compression, and Elevation).

**Quick Quiz**

- What is the concern with distal humerus fractures?
- What is compartment syndrome?
- Know subluxation of the radial head!
- Name the common physical findings in a sprain of the ankle.
- How do you treat an ankle sprain?
- What are the symptoms of a corneal abrasion? How do you diagnose it?
- What is hyphema?

Ice for 20 minutes every 2 hours for 48 hours to prevent swelling. The patient should remove the ice if contact-area numbness develops and protect the skin with a towel if using a plastic bag! Severe strains require a splint for protection and comfort.



Courtesy of Thomas Kyzmarick, MD

*Image 3-8: Sprained Ankle*

**Table 3-3**: Grade of Ankle Sprains

| | Grade I (Mild) | Grade II (Moderate) | Grade III (Severe) |
|---|---|---|---|
| Swelling | Mild | Moderate | Severe |
| Tenderness | Mild | Moderate | Severe |
| Loss of function | Minimal | Difficult to ambulate | Unable to bear weight |
| Treatment | 7–10 days rest | 2–4 weeks rest | 5–10 weeks rest |

## EYE ABNORMALITIES

### CORNEAL ABRASION

Symptoms of corneal abrasions include pain, tearing, photophobia, and decreased vision. Diagnosis is best made with fluorescein dye and a slit/Wood's lamp (Image 3-9). An abrasion is transparent, whereas an ulcer is opaque; both light up under fluorescein. Treatment with a topical antibiotic is acceptable.

Do not send the patient home with a topical anesthetic; use these in the office only. Also, do not use a semi-pressure patch or miss a foreign body.

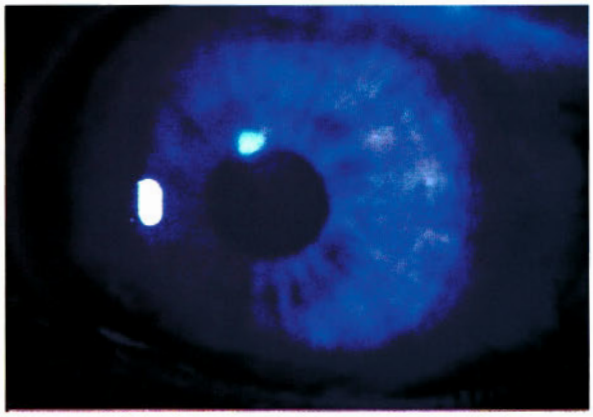
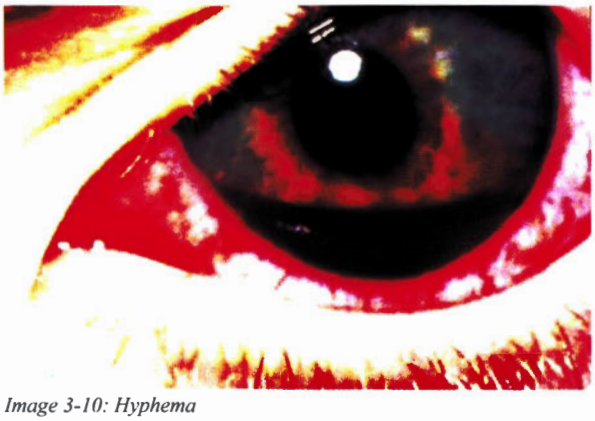


*Image 3-9: Corneal Abrasion with Ulceration*

### HYPHEMA

Hyphema (Image 3-10) is the presence of blood in the anterior chamber of the eye. Usually, it occurs after a blunt or perforating injury. It appears as a bright or dark red fluid level between the cornea and iris or a diffuse murkiness of the aqueous humor. Eye pain is common, as is somnolence. Ophthalmologic consult is required.

Treatment includes topical steroid and cycloplegia drops, as well as a protective eye shield and bed rest, with the head elevated 30–45° and close monitoring of intraocular pressure.



Courtesy of Thomas Kyzmarick, MD

*Image 3-10: Hyphema*

# PALS

## OVERVIEW

Make sure you know everything on these last few pages about PALS (Pediatric Advanced Life Support). Included are only the main areas that would likely appear on the Board exam.

Always start with your ABCs. For A, remember head tilt/chin lift or, if trauma is present, jaw thrust. Clear the airway of foreign bodies via back blows and chest thrusts for children < 1 year. For those 1–8 years, use the Heimlich maneuver.

For B, don't forget supplemental oxygen (if available). For infants, use mouth-to-mouth/nose breathing. For children, pinch the nose and use mouth-to-mouth breathing. Breaths are delivered slowly over 1–1½ seconds.

For C, we'll review in great detail below.

For newborns, remember the ABCs and temperature (warm and dry). Use the inverted pyramid, which outlines the resuscitation that is often needed by newborns (position, clear airway, stimulate by drying, give oxygen as needed), as well as the procedures needed less frequently (establish ventilation with bag and mask) and those needed rarely (chest compressions, medications).

## CIRCULATION

### Overview

Where should you do a pulse check? Newborns: umbilical; infants: brachial or femoral; children: carotid pulse. Start compressions for HR < 60 with poor perfusion (Table 3-4).

### Pediatric Tachycardia

Covered in the Cardiology section.

### Pediatric Bradycardia

Possible causes of bradycardia include hypoxemia, hypothermia, head injury, heart block, heart transplant, and toxins.

For bradycardia, manage ABCs, give oxygen, and monitor. If there is no cardiovascular compromise (abnormal vital signs, altered consciousness), just observe, support ABCs, and consider transfer.

For cardiovascular compromise (e.g., low BP) and HR < 60:

- Perform chest compressions.
- Epinephrine: (May repeat every 3–5 minutes at this dose.)
  - 0.01 mg/kg (1:10,000, 0.1 mL/kg) IV/IO
  - 0.1 mg/kg (1:1,000, 0.1 mL/kg) endotracheal tube
- Atropine: Give atropine first if you suspect increased vagal tone or primary AV block!
  - 0.02 mg/kg, minimum of 0.1 mg
- Consider pacing.

### Pediatric Pulseless Algorithm

Possible causes of pulseless arrest include the 5 H's (hypoxemia, hypothermia, hypovolemia, and hypo- or hyperkalemia), metabolic disorders, and the 4 T's (tamponade, tension pneumothorax, thromboembolism, toxins).

For a pulseless arrest, manage ABCs, give oxygen, and monitor. If there is still no pulse, assess with an ECG.

**Table 3-4:** Pediatric CPR

| CPR | Infant < 1 yr | Child 1 yr to Adolescent | Adolescent |
|---|---|---|---|
| Airway | Head tilt/chin lift (jaw thrust if trauma) | | |
| Obstructed airway | Back blows and chest thrusts | Abdominal thrusts | |
| Breathing | 2 initial breaths | | |
| Breathing: No CPR | 12–20 breaths/min | | 10–12 breaths/min |
| Breathing and CPR | 8–10 breaths/min | | |
| Circulation | Brachial/femoral | | Carotid pulse check |
| CPR technique | 2 thumbs with encircling hands at lower half of sternum | Heel of one hand: other hand may be on top | |
| Rate | 100/min | | |
| Ratio | 15:2 | | 30:2 |

**QuickQuiz**

- Explain how to do rescue breaths in an infant and a child. How are they different for each?
- Know Table 3-4.
- How do you manage symptomatic bradycardia?
- How do you manage pulseless V-tach?

For a pulseless child, if the ECG shows V-fib or V-tach:

- SHOCK: Attempt defibrillation.
  - Up to 3 times
  - Dose: 2 J/kg, 2–4 J/kg, 4 J/kg
- Then, pattern should be DRUG→ CPR→ SHOCK (repeat).
- DRUG—Give epinephrine every 3–5 minutes.
  - 0.01 mg/kg (1:10,000, 0.1 mL/kg) IV/IO, or
  - 0.1 mg/kg (1:1,000, 0.1 mL/kg) endotracheal
- CPR
- SHOCK – i.e., attempt defibrillation.
- Use 4 J/kg within 30–60 seconds of every medication.
- Consider use of other drugs: amiodarone, lidocaine, magnesium (for *torsades de pointes* or hypomagnesemia).

If pulseless and the ECG shows anything other than V-fib or V-tach (e.g., PEA, asystole) drop SHOCK from the pattern:

- CPR
- DRUG: Give epinephrine every 3–5 minutes.
  - 0.01 mg/kg (1:10,000, 0.1 mL/kg) IV/IO, or
  - 0.1 mg/kg (1:1,000, 0.1 mL/kg) endotracheal
- Continue CPR up to 3 minutes.
- Reassess ECG.

MedStudy

PEDIATRICS BOARD REVIEW

PEDS

**CORE CURRICULUM**

5th EDITION

Authored by J. Thomas Cross, Jr., MD, MPH, FAAP
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# ADOLESCENT HEALTH AND GYNECOLOGY

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# Adolescent Health and Gynecology

# SPECIAL ISSUES OF ADOLESCENTS

Adolescents are a distinctive group of individuals. The Boards like to ask about them because of the ways they differ from younger children. The definition of an adolescent varies, but most consider adolescents to be 10–21 years of age. Things to note:

Adolescents receive much of their care in emergency rooms, and much of this is "non-urgent" care.

Injuries are the leading diagnostic category.

Females present frequently with abdominal pain, menstrual irregularities, or sore throat as their main complaint.

In the general outpatient clinic, health supervision is the most common diagnostic code for 10–14-year-olds. For 15–24-year-olds, the most common diagnostic code is pregnancy! Acne is the 3rd most common problem presented.

The reasons for hospitalizations for adolescents (10–21-year-olds) in order of incidence:

1) Pregnancy (3% of all pediatric hospitalizations!)
2) Emotional disorders
3) Injuries
4) Digestive tract disorders
5) Respiratory tract diseases

Smoking, alcohol, and illicit drug use are continuing problems, and obesity is an emerging issue.

Automobile and motorcycle accidents are the leading cause of adolescent morbidity and mortality. Homicides are the 2nd leading cause of death and the #1 cause of death in African-American adolescents. Suicide is the 3rd leading cause of death.

Remember these items when a Board question refers to an adolescent. Such questions are likely to involve sex, drugs (even rock and roll if hearing loss is an issue!), injuries, mental illness, pregnancy, emancipation rights, or supervisory guidance.

# THE DEVELOPING ADOLESCENT

## GENERAL CONSIDERATIONS

Many define the beginning of puberty as the beginning of adolescence. Multiple studies have been published with, unfortunately (for you the test taker), varying ages. What I give you here is the most recent data and that supported by Nelson's *Textbook of Pediatrics*. In the U.S., pubescence occurs in Caucasian girls at a mean age of 10.0–10.4 years (7.8–11.6) and for African-American girls at a mean age of 8.9–9.5 years (6.1–10.1). Puberty for girls generally lasts an average of 4 years (1.5–8 years). In recent years, the mean age has been reported at about 9.5 years for African-American boys and 10 years for Caucasian boys. For boys, puberty usually lasts an average of 3 years (2–5 years).

Puberty affects everything! Skeletal growth and body composition, as well as cardiorespiratory, hematologic, neuroendocrine, and reproductive changes occur at varying rates and stages. Chronological age frequently does not correlate with biological maturity. Because of this, most use sexual maturation ratings (our old friend Tanner) to provide us with guidance regarding the biological maturation of adolescent patients—these are covered in great detail in the Endocrinology Section.

Skeletal Growth: During adolescence, skeletal growth accounts for ~ 25% of final adult height. The growth spurt for girls occurs earlier than for boys (Tanner 2–3 for girls vs. Tanner 4 for boys). Girls also reach their final adult height earlier than boys (average: 16 years of age for girls and 18 years of age for boys). Hint: Just think of the tall high-school girl with the short high-school boy!

Body Composition: 40% of adult weight is gained during adolescence. In boys, lean body mass actually increases from 80% to 90%, but girls see a decrease from 80% to 75%. Body fat changes also differ between girls and boys. Body fat increases up to 26.7% in girls throughout puberty. Body fat increases up to 11.2% in boys, occurring very early in puberty and remaining stable thereafter. Visualize the chubby 12-year-old boy at the beach who, at 17, is lean and mean. During puberty, girls keep adding adipose to appropriate areas for "curves" to occur. Muscle mass generally peaks after the growth spurt.

Hematologic Changes: In boys, blood volume, RBC mass, and hematocrit all increase during puberty. No corresponding increases occur in girls.

Cardiorespiratory Development: The heart doubles in weight and systolic blood pressure increases for boys, but these remain stable for girls. For both boys and girls, vital capacity increases markedly as lung size increases; additionally, respiratory rates fall while heart rates increase.

Neuroendocrine Changes: As any parent can tell you, the structure and mass of the brain do not change during adolescence, but there are definite changes in brain activity! It appears that adolescents gradually develop the α rhythm of the adult brain.

Reproductive System Changes: These will be discussed in greater detail in the Endocrinology section—including pictures of the Tanner stages. Girls usually show signs of the onset of sexual maturation with breast development (thelarche), which occurs anywhere between ages 6 and 11.5 years. The mean age for the beginning of menses is 12.5 years (10–16.5 years). For boys, testicular growth and thinning of the scrotum are the first signs of puberty. Penis lengthening is followed by growth of pubic hair. Axillary hair occurs around mid-puberty.

## SURVIVING ADOLESCENCE

Here, we get into those "difficult" years and their attendant issues. Androgens are probably responsible for many of the behavioral changes seen in adolescents. Boys have increasing conflict with their parents, particularly with their mothers, and especially during the growth spurt. Also, all of the extra testosterone causes them to behave more impatiently, aggressively, and irritably. Girls have increased conflicts with their mothers and less contact with their fathers.

On the Board exam (and in the real world), look for a girl maturing early who likely has greater dissatisfaction with her looks, lower self-esteem, and a greater degree of unhappiness. These girls tend to manifest problems in school as their academic interests wane. The opposite is true for boys—it's the "late bloomers" who tend to have a more negative self-concept and body image.

There is a behavioral spectrum that all adolescents go through that requires closer examination.

Early Adolescence (10–13 years): At this stage, often characterized by preoccupation with self, the preteen has lots of questions about physical maturation and engages in occasional masturbation. There is an increasing emphasis on relationships with peers, testing of authority, and less interest in parental relationships. Cognition is concrete! (For the Boards, if a health issue is presented relative to this age period, look for an answer option favoring the use of simple, explicit discussions using visual and verbal cues.) Kids in this age group tend to be impulsive.

Middle Adolescence (14–16 years): Pubertal development is nearing completion, and these kids explore relationships with the opposite (and same) sex. Masturbation frequency increases. Their peers are the most important group for support and effecting change. Cognition begins to mature, but there is noticeable immaturity in their ability to interact with the real world. Feelings of omnipotence and immortality often lead to risk-taking and impulsive behaviors.

Late Adolescence (17–21 years): Body image and gender role are defined. Individual relationships become more important than peer group. Idealistic! Cognitive development is complete. "Life goals" become the center point of discussion. You will still see impulsiveness, but now with the cognition to understand their actions and a greater ability to delay gratification and make independent decisions. (Well, sometimes!)

## EMANCIPATION AND HEALTH CARE

This is a difficult one for the Boards. In the U.S., the right of an adolescent (minor < age 18 years) to seek and receive treatment without parental consent varies from state to state. Usually, public health statutes specify the right to self-consent for treatment when there is clinical suspicion of a sexually transmitted disease.

Many states allow minors to seek help for pregnancy, contraception, drug and other substance abuse, and mental health issues without parental consent. This could be a problem for the Boards—each state has different laws, but for most questions in this area, they go with the "majority of states." So be careful with your personal views or the laws in your particular state. For many of you, these questions are difficult. However, there are some "absolutes" or "almost absolutes":

- Emancipated minors: These kids have moved outside of the home and pay their own bills, etc. They are no longer subject to parental authority. Also, this category includes those who are married or members of the military. In most states being a parent also makes you an emancipated minor.
- Emergencies: The physician's judgment to treat may occur without explicit consent of the parent (or the patient, for that matter) if the patient is unconscious or unable to give consent.
- Mature minor concept: This is relatively recent. There is a trend toward recognizing minors who have the ability to comprehend the risks and benefits of a specified treatment/ therapy, and they may provide their own consent. Usually, this occurs in cases where the care is "low risk" and will clearly benefit the minor.

Questions about the adolescent making treatment decisions/choices are likely to revolve around a mature minor who understands the risks and benefits of treatment and how much of a role they should have in making decisions. The answer: Their role in decision-making is commensurate with their level of maturity, not their chronological age!

Confidentiality is a whole "nuther can of worms." Generally, if you have the ability to self-consent on an issue, you have the right to confidentiality. Exceptions to remember (especially for the Boards) are child abuse and cases where the minor may cause harm to self or someone else, in which case the physician must notify parents or appropriate public/private officials. Some states require legal disclosure to parents, depending on the diagnosis, and this requirement must be conveyed to the minor. The best choice is to encourage the minor to agree to bring the parents or guardian into the decision-making process, with you acting as facilitator.

Speaking of confidentiality, what about parents who ask for drug testing of their adolescent without the adolescent's consent? The AAP says this is only OK in 2 situations:

1) If the adolescent lacks decision-making capacity, or
2) If the information obtained on history and physical is strongly concerning for a substance abuse problem

**QuickQuiz**

- What is the most common reason for 10–14-year-olds to visit outpatient clinics? 15–24-year-olds?
- What is the #1 reason for hospitalizing adolescents?
- What is the mean age for puberty in Caucasian girls? African-American girls?
- True or false? Boys have their pubescence growth spurt before girls.
- Define an emancipated minor.
- What are the medical exceptions for notifying parents against an adolescent's wishes?
- What parts of the physical examination are important to focus on in an adolescent boy or girl?

Other correct responses on the Board to be aware of if this issue comes up:

- Talk to the parents to understand why they think this test is needed.
- Talk to the parents on the limitations of drug testing and more appropriate methods of detecting substance use; e.g., history and physical.
- Talk to the parents about the harms of the test (violating the trust between parent and adolescent, and between physician and adolescent patient).

## THE ADOLESCENT HEALTH VISIT

The key here is to allow the adolescent to become autonomous, and to involve the parents only as much as the adolescent wishes. For matters involving sex and substance use, interview the adolescent alone. However, inform your patient that in areas of life-threatening behavior (e.g., suicide, management of chronic disease), you have the right to (and by law, must) involve a parent or other guardian.

Psychosocial assessment is very important in this age group but frequently difficult to ascertain. Ask questions about peer and family relationships; depression; sexual relationships, if they have occurred; substance use; and eating disorders, as described in Table 4-1. Understand that a minor's chief complaint (common example, "sore throat") at presentation may not be the real reason for the patient's visit, and it may require a follow-up visit to determine the true problem.

For this visit, the AAP recommends discussion (using non-judgmental, open-ended questions) about normal development, home environment, risk of substance abuse, screening where appropriate for physical/sexual abuse, academic performance, peer relationships, injury prevention, nutrition and dietary habits, physical activity, dental health, breast or testicular self-examination, and skin protection. Recently, the AAP added recommendations to discuss "online behavior" and to counsel on bullying.

Focus physical examination on these important areas:

- Height/weight/vital signs
- Hearing testing, especially if the adolescent admits to listening to loud music
- Vision testing, because the growth spurt also affects the globe of the eye, possibly resulting in myopia
- Screening for hypertension
- Scoliosis, which occurs in 5% of boys and 10–15% of girls (The cutoff for referral to orthopedics is generally a 10° curvature or greater.)
- Skin: acne, warts, fungal infections, suspicious moles
- Sexual maturity rating
- Breast examination (Most recommend teaching about breast self-examination in this age group—even though the risk of breast cancer is near zero—because of the potential to start "good habits" early. Note: This is extremely controversial. The current 2010 USPSTF recommends against breast self-exam for all women; the American Cancer Society guidelines disagree aggressively with this and state that they should always be done—both self- and clinical breast exams.)
- Pelvic examination, primarily if the girl is sexually active or is having menstrual problems/abdominal pain

**Table 4-1:** Obesity and Eating Disorders—Recommended Milestones for Intervention

| |
|---|
| **Obesity** |
| Body Mass Index (BMI) ≥ 95th percentile |
| OR |
| BMI between 85th and 95th percentile |
| AND |
| Family history of premature heart disease, obesity, HTN, or DM |
| HTN |
| Cholesterol > 200 mg/dL |
| Increase of ≥ 2 points in BMI in 12 months |
| Adolescent is concerned about his/her weight |
| **Eating Disorders** |
| Weight loss > 10% of previous weight |
| Recurrent dieting when not overweight |
| Adolescent discusses distorted body image |
| BMI < 5th percentile |

- Scrotum examination (It is very important to instruct boys in self-examination since testicular cancer does occur in this age group!)
- Obesity and other eating disorders (Recommend further assessment based on elements in Table 4-1.)

Laboratory screening is much more controversial and is not as well documented as necessary. However, screen any sexually active adolescent for sexually transmitted diseases (STDs). Conduct a pelvic exam with Pap smear for those ≥ 21 years of age. This is the new 2011 ACOG recommendation, which is different from the current AAP recommendations—but likely is acceptable. (The older recommendations suggested Pap for all sexually active girls regardless of age.) The reason for the guideline changes is because sexually active adolescents are more likely than adult women to become infected with HPV (almost always noncancer serotypes) and to have abnormal cervical cytology screening; however, these are usually transient, and cervical cancer is exceedingly rare in young women. Treatment for abnormal cytology, that is likely not due to a "cancer-causing" HPV type, has long-term effects. Thus, the benefits of adolescent screening may be offset by potential harms, weighing against aggressive screening in this age group. This is why HPV DNA testing is not recommended in women < 30 years of age as a screening tool, because many of them will be HPV-positive with noncancerous serotypes—thus resulting in unnecessary testing and worry.

Newer recommendations specifically recommend screening all asymptomatic, sexually active females for *Chlamydia*. The CDC suggests using nucleic acid amplification tests (NAATs) on urine specimens to screen for both gonorrhea and *Chlamydia*. Vaginal swabs (NAATs) now are commonly used and can even be self-collected (in office). Remember that HIV testing is now universally recommended by the CDC for all who are sexually active. In addition, the AAP recommends annual dipstick urinalysis for leukocytes for all sexually active males and females; however, the use of dipstick leukocyte screening is not recommended by "Bright Futures," an educational center associated with the AAP, or by the USPSTF. Only the AAP recommends a routine urinalysis at least once during adolescence. To add to the confusion, remember that up to 1/3 of healthy adolescents have small amounts of proteinuria.

All menstruating girls are at risk for anemia, but guidelines differ on what to do. The AAP recommends at least one measurement of Hgb or Hct for all menstruating adolescents, preferably at age 15; however, "Bright Futures" recommends only selective screening in adolescents at increased risk, including athletes and vegetarians, as well as those who are underweight/malnourished, have chronic illness, or have a history of heavy menses (defined at > 80 mL/month).

Bright Futures recommends a fasting lipid profile once in adolescents—between the ages of 18 and 21 years. And then consider cholesterol screening for those < 18 years of age with a family history for early cardiovascular disease (parent or grandparent < 55 years of age) or hyperlipidemia (parent with serum cholesterol ≥ 240 mg/dL). Most recommend tuberculin skin tests for youth who are at risk (e.g., immigrants from a region with high incidence of tuberculosis; IV drug use; history of incarceration or volunteer work at a prison; those who are homeless, work in a health care facility, or have HIV), but not for general screening. (This is known as "targeted screening.")

Immunizations [Know]: If the adolescent has not had 2 MMRs, give the second one in early adolescence by age 12 years. Tdap is due at age 11–12 years, then Td is due every 10 years thereafter. Also, give the hepatitis B immunization in early adolescence if the teen hasn't already received it. Consider giving the varicella vaccine, if the patient has not been vaccinated and there is no reliable history of chicken pox (although some recommend checking titers before giving the required 2 immunizations). Hepatitis A vaccine is indicated for all children in the U.S., particularly for youth who are high risk or already have chronic liver disease. The 3-dose HPV vaccine series is currently recommended for all girls as early as 9 years of age (HPV4 or HPV2). HPV4 can be given to males after age 9 years to prevent genital warts. Meningococcal conjugate vaccine (MCV4) is recommended at 11–12 years with a follow-up booster dose at 16 years. Yearly influenza vaccine is now recommended for all children. PCV13 (conjugate pneumococcal vaccine) is recommended for adolescents with chronic cardiovascular or pulmonary disease and with other conditions; e.g., nephrotic syndrome, hemoglobinopathy, asplenia, and HIV infection. These patients should also receive the 23-valent pneumococcal vaccine (optimally 8 weeks after receiving PCV13). A single PPV23 revaccination should be administered after 5 years to children with functional or anatomic asplenia or an immunocompromising condition.

It is very important that you provide parents or other adult caregivers health guidance at least once during their child's early, middle, and late adolescence.

Include in this information:

- Normal adolescent development
- Signs and symptoms of disease and emotional distress
- Parenting behaviors that promote healthy adolescent adjustment
- Ways to be role models for adolescent behaviors (driving, alcohol use, etc.)
- How to monitor/manage new drivers
- Adequate weapon control in the house
- Removing weapons and lethal medications in a house where an adolescent has suicidal intent
- Ways to monitor adolescent social and recreational use of tobacco, alcohol and other drugs, and sex
- Appropriate social relationships

**QuickQuiz**

- How many MMR inoculations should adolescents have by the time they are 12?
- How often should adolescents see a medical professional for health supervision and guidance?
- Who is more likely to smoke—a boy or girl? Drink?

A key point that the Boards might ask: To promote injury prevention, give adolescents health guidance annually—including seat-belt use, alcohol/substance/tobacco use (especially while driving), helmet use, violence management, weapon safety, and exercise preparedness to prevent injury). Most also recommend annual diet and exercise guidance, as well as health guidance regarding sexual behaviors and substance abuse (including anabolic steroids). The key thing to know here is that if adolescents participate in one risky behavior, it is more than likely they are participating in others!

For those with chronic conditions, begin the discussion in early adolescence about transition of their care to an adult provider, and help facilitate this as a smooth transition with the child and parents. For visits that require new or old medications, discuss the importance of adherence.

Much of the above comes from the Guidelines for Adolescent Preventive Services (GAPS).

## SUBSTANCE ABUSE

Alcohol and tobacco use remains alarmingly high in adolescents, but tobacco use has trended downward over the last decade. The mean age for smoking is 12 years, and it is 12.6 years for alcohol consumption. Girls smoke more often than boys (but a recent report shows that boys = girls), and boys consume alcohol nearly twice as often as girls. About 75% of 12$^{th}$-grade adolescents reported "ever drank alcohol" and 36% of high school seniors report that they have had ≥ 5 drinks in a row in the last 30 days!

Around 33% of current high school seniors admit to using marijuana within the previous year. The mean age of first use is 14.4 years. Around 7% of adolescents have ever tried cocaine, 6% have ever tried MDMA (ecstasy, X), 4% have used inhalants (paint thinners, hair spray, "poppers", glue, shoe polish, spray paint) within the previous 30 days, 3% have ever tried meth, and 1.5% have ever tried heroin.

How can you tell if a kid is on drugs? Signs vary and can be very difficult to interpret [Know]:

- An increasing degree of emotional and/or physical isolation from family
- Absent or hostile communication
- A decrease in school performance or increased absenteeism
- New disinterest in athletics
- Changes in peer group population
- Involvement in a crime
- Participating in other risky behaviors, including sex

Risk factors for development of substance abuse are listed in Table 4-2.

Now a more controversial question: When is it appropriate to screen an adolescent for substance abuse? The AAP's policy is that drug testing of the older, competent adolescent should be voluntary. Parental consent alone is not sufficient. However, look for certain signs that make you think about screening (Table 4-3). Almost all drugs can be routinely tested in urine. The one exception is LSD (lysergic acid diethylamide). The maximum window of detection following the most recent time of drug ingestion for the majority of drugs of abuse is 24–72 hours, with 2 exceptions: PCP metabolites may be detected for up to 8–10 days and marijuana metabolites may be detected for up to 4–6 weeks in chronic daily users. See Table 4-10 and Table 4-11 at the end of this section for commonly abused substances and their side effects/clinical manifestations.

**Table 4-2:** Risk Factors for Development of Substance Abuse

| |
|---|
| Household drug use (especially by parents) |
| Peer group drug use |
| Attention deficit disorder (ADD, ADHD) |
| Depression |
| Anxiety disorder |
| Impulse control problems |
| Unnecessary, aggressive outburst |

**Table 4-3:** Substance Abuse Testing—Reasons to Screen in Adolescent

| |
|---|
| Trauma |
| Unexplained accident |
| Psychiatric symptoms |
| School performance deterioration |
| Unexplained chronic illness |
| Increased absenteeism |
| Suicide attempt |
| Altered mental status |

Recently there have been huge increases in adolescent abuse of prescription (ADHD meds, not necessarily their own) and over-the-counter medications (dextromethorphan, etc.).

## DEPRESSION

In prepubertal children, there is no gender difference in rates of depression. However, at postpuberty, girls are 2–3x more likely than boys to experience depression. Comorbid mental illness is common; and don't forget to look for anxiety disorders, substance abuse, attention-deficit disorder (ADD), and disruptive behaviors (oppositional-defiant or conduct disorders). Unfortunately, 70% of depression in adolescents is un- or under-recognized and goes untreated.

Look for 9 symptoms, in addition to depressed mood/irritability but particularly diminished interest or lack of pleasure in all previously enjoyable activities (especially on the Board exam!) (Table 4-4). If an adolescent has the major symptoms of depressed mood/irritability, 4 or more of the 9 other findings in Table 4-4, and these symptoms occur for > 2 weeks, a "major depressive disorder" is occurring.

Note: If these symptoms occur within 3 months of an identifiable stressor, consider them part of an adjustment disorder with depressed mood. Also, don't forget that, in adolescents, depression can present as sadness or boredom.

Dysthymic disorder presents as a more chronic depressed state with at least 3 of the 9 symptoms present for at least 1 year, without evidence of specific major depressive episodes.

Bipolar affective illness, or cyclothymic disorder, occurs when the adolescent has periods of depression alternating with mania or hypomania, which may be characterized by intense energy, sleeplessness, racing thoughts/pressured speech, grandiosity, and excessive/impulsive risk-taking behaviors.

If you suspect depression, also consider other abnormalities as etiologies for the symptoms/signs: hypothyroidism, nutritional deficiencies, chronic infections (e.g., mononucleosis, HIV—if at risk), or a chronic systemic illness (e.g., SLE). Don't forget that substance abuse frequently presents with depression symptoms/signs.

**Table 4-4**: Symptoms of Depression in Adolescents

| |
|---|
| Weight loss, gain, or failure to make expected weight milestones |
| Decrease or increase in appetite |
| Insomnia or hypersomnia |
| Psychomotor agitation or retardation |
| Fatigue and energy loss |
| Feelings of worthlessness |
| Excessive/inappropriate feelings of guilt |
| Decreased ability to concentrate/think |
| Recurrent thoughts of death |

Treatment for depression is complex and requires a multidisciplinary approach that requires psychosocial treatments, including individual and family therapy, cognitive behavioral therapy, and medications such as SSRIs (selective serotonin reuptake inhibitors). Fluoxetine has the most evidence of efficacy in pediatric patients. Recent literature, however, has called into question the "overuse" of SSRIs in children and adolescents, especially when other prior therapies have not been attempted. Additionally, recent reports show an increased risk (from 2% for placebo to 4% for treatment arms) of suicidal thoughts and behaviors (but not completed suicides) in children and adolescents on SSRIs. These reports caused further controversy after the FDA directed "black box" warnings stating that use of all antidepressants in children and adolescents increases the risk of suicidal ideation and behavior.

Head-to-head studies demonstrate that tricyclic antidepressants are not as effective as SSRIs for early-onset depression, and are associated with significant morbidity and mortality following intentional overdose; so avoid them as treatment. Once started, continue SSRIs for a minimum of 6 months.

Depression may be the first presentation of bipolar disorder. Risk factors for bipolar disorder in a depressed adolescent include rapid onset of depressive symptoms, associated psychotic symptoms, and a family history of bipolar disease. Bipolar disorder requires therapy with lithium, valproic acid, or carbamazepine. Although it is controversial, bipolar disorder is also frequently treated with a mood stabilizer or antipsychotic.

The earlier the adolescent/child is affected with depression, the more severe and recurrent the depression is likely to be. If untreated, a major depressive episode can last 7–9 months; recurrence occurs in 70% of adolescents within 5 years.

When do you hospitalize adolescents with depression? Hospitalization is appropriate when the patient is suicidal or homicidal, psychotic, manic, or abusing substances, and is not responding to outpatient therapy.

## EATING DISORDERS

### Overview

The incidences of anorexia nervosa and bulimia have increased in recognition and occurrence in the last century. There appears to be a genetic predisposition for eating disorders, and serotonin activity in particular may play a role. Neurotransmitters may also play an etiologic role in development of these disorders.

According to a December 2010 AAP clinical report on eating disorders, 0.5% of all adolescent girls have

**QuickQuiz**

- Name 7 of the 9 symptoms to look for in depression.
- Under what conditions should you hospitalize an adolescent with psychological problems?
- How common is anorexia nervosa?
- What are the diagnostic criteria for anorexia nervosa?
- In anorexics, what ECG changes suggest increased risk for ventricular arrhythmias?

anorexia nervosa, 1–2% bulimia, and males make up 5–10% of eating-disorder patients. (There is definitely an increasing incidence in males.) In addition, the prevalence of eating disorders (not otherwise defined) is anywhere from 0.8–14% because many adolescents do not meet DSM-IV criteria.

## Anorexia Nervosa

Anorexia nervosa occurs in about 1/100 girls 16–18 years of age. The peak incidences usually occur at 2 points in time: ages 14.5 years and 18 years. These peaks correlate with times when affected patients change schools (junior high to high school, etc.). Some estimates say 25% of these girls may start an eating disorder before age 13! The 2010 AAP clinical report showed that hospitalizations for eating disorders increased 119% in children < 12 years of age!

Girls outnumber boys by 10:1 for this diagnosis. One source suggests that affected boys may also have a higher incidence of "gender identity" issues.

Diagnose anorexia nervosa by using the DSM-IV criteria (Table 4-5). Often, girls appear "normal" before exhibiting this disorder; yet, in reality, they tend to be overachievers and perfectionists. Other psychiatric disorders are commonly associated, including obsessive-compulsive and affective disorders. Risk periods seem to be during the transitions into middle/junior high, then high school and college. Also, these disorders commonly are preceded by an announced intention to go on a diet. On the Boards, look for the gymnast, ballet dancer, long-distance runner, or the male wrestler!

**Table 4-5:** DSM-IV Criteria for Diagnosis of Anorexia Nervosa

| |
|---|
| Intense fear of becoming obese, which does not decrease as weight loss occurs |
| Disturbance in the way the adolescent views her/his own body weight, size, or shape (examples: "I feel fat" even though normal or abnormally below weight for height; "My thighs are huge" even though no body fat is visible) |
| Refusal to maintain body weight over a minimal normal weight for age and height (15% below the norms for weight for height) |
| In females, absence of 3 consecutive menstrual cycles |

Early intervention is critical. Conduct a general physical exam, including body mass index, vital signs, and orthostatic measurements. Physical findings may include excessive lanugo hair; dry, hyperkeratotic skin; acrocyanosis; dependent edema; and mitral valve prolapse. In severe anorexia, heart rates may fall to 20–40 beats/min, and hypothermia is fairly common. Consider lab tests if the patient has been abusing laxatives and/or diuretics, or inducing vomiting (look for enamel destruction on the teeth from the acid). With laxative and diuretic abuse and excessive vomiting, look for hypokalemia and a hypochloremic metabolic alkalosis. Hematology can also be severely affected, with bone marrow suppression resulting in low WBC counts, thrombocytopenia, decreased ESR, and anemia. In girls, LH, FSH, and serum estradiol levels are low. In boys, testosterone levels are low. In both boys and girls, cortisol and endorphins are elevated. Chemistry abnormalities include elevated serum transaminases and increased levels of BUN, cholesterol, and carotene.

Osteopenia (now becoming more commonly known in younger patients as "decreased bone density for age") is one of the most severe complications of anorexia nervosa; therefore, daily intake of 1,200–1,500 mg per day of elemental calcium and a multivitamin that provides 400 IU (some recommend up to 1,000 IU) of vitamin D is recommended. Dual-energy x-ray absorptiometry (DXA) scanning is frequently indicated to monitor for evidence of osteopenia.

ECG changes: Look for ST-segment depression on exercise stress testing or prolonged QT intervals—these changes are associated with increased risk of ventricular tachycardias! Bradycardia and low-voltage changes are common. U-waves are consistent with hypokalemia. CHF can occur if the severely debilitated patient is hydrated too rapidly. The risk of heart failure is greatest in the first 2 weeks of refeeding; therefore, limit daily weight gains to 0.2–0.4 kg/day. Echocardiogram may show ventricular wall thickness, mitral valve prolapse, and pericardial effusion.

Anorexia nervosa has been associated with COPD-like changes on chest x-ray. But the clinical significance of these changes is not known because pulmonary function tests are generally normal.

Amenorrhea is also common. Due to the hypoestrogenic state, withdrawal bleeding does not occur if progesterone is given. Menses return after achieving 90% of ideal body weight.

Treatment is comprehensive and involves psychotherapy (for both the patient and family), behavior modification (cognitive behavioral therapy), and nutritional guidance.

| Table 4-6: Anorexia Nervosa—Criteria for Hospitalization |
|---|
| < 75% IBW or ongoing weight loss despite intensive management |
| Refusal to eat |
| Body fat < 10% |
| HR < 50 daytime, < 45 sleeping |
| SBP < 90 |
| Orthostatic changes: > 20 pulse, > 10 in blood pressure |
| Temperature < 96° F |
| Arrhythmia |
| Suicidal ideation |

Consider pharmacotherapy if depression is also present. Additionally, SSRIs may be helpful in preventing recurrence of anorexia but have not been shown to be helpful in the actual treatment of anorexia. Table 4-6 lists criteria for hospitalization. Be aware that these patients are at risk for refeeding syndrome and electrolyte abnormalities during treatment:

- Hypophosphatemia—know this one!
- Hypokalemia
- Hypomagnesemia
- Vitamin (e.g., thiamine) and trace mineral deficiencies
- Volume overload and edema

### Bulimia Nervosa

Bulimia nervosa is even more common than anorexia nervosa—it occurs in 1–2% (according to 2010 AAP clinical report, but previous studies mention up to 3–5%) of girls but < 1% of boys. Adolescents may have anorexia and bulimia concurrently, or may alternate between the two. Generally, bulimics are older girls in the mid-to-late adolescent period. When compared to individuals with anorexia, they are more outgoing, impulsive, and prone to acting-out behaviors, including stealing, sexual promiscuity, and self-destructive acts. The goal of the bulimic is to lose weight. Bulimia is also a DSM-IV diagnosis (Table 4-7). These adolescents may be of normal weight or slightly overweight. Initial symptoms may include fatigue, bloating, irregular menses, and chest pain accompanied by sore throat (due to excessive vomiting). Physical findings are important, especially on the Board exam! Look for edema (fluid retention), bilateral painless parotid gland swelling, calluses on the dorsum of the fingers (also known as Russell signs) from inducing vomiting, and loss of tooth enamel from the acidic vomit. Aspiration pneumonia can also occur. If the patient is using syrup of ipecac, she may induce hypokalemia and resultant cardiac toxicity. Also look for a metabolic alkalosis and elevated amylase levels from the recurrent vomiting.

| Table 4-7: DSM-IV Criteria for Diagnosis of Bulimia |
|---|
| Recurrent episodes of binge eating (rapid intake of food in a short period of time) |
| A feeling of "lack of control" over eating behavior during these binges |
| Regularly has self-induced vomiting; uses laxatives, enemas, or diuretics; strict dieting or fasting; or vigorous exercise to prevent weight gain |
| A minimum average of 2 binge-eating episodes/week for at least 3 months |
| Persistent concern with body shape/weight |

Bulimia often accompanies an obsessive-compulsive or affective disorder. Consider SSRIs in this subset of patients. Interventions must address the binging/purging behavior early on, as well as the underlying psychopathology. Pharmacologic agents are much more effective in bulimia than in anorexia. Fluoxetine has shown the best results. Criteria for hospitalization are listed in Table 4-8.

### Binge Eating Disorder

Binge Eating Disorder is a newly recognized disorder characterized by recurrent episodes of binge eating in a discrete period of time associated with a sense of lack of control. Rapid intake of large amounts of food when not physically hungry usually occurs when the individual is alone and is followed by feeling depressed, embarrassed, and disgusted with oneself. Binge eating occurs at least twice weekly for 6 months or more and is not associated with the regular use of inappropriate compensatory behaviors (e.g., purging or excessive exercise).

## SEXUAL DEVELOPMENT

### Female Issues

#### Breast

Estrogen is the most influential factor in breast development during puberty. It binds to breast tissue and causes

| Table 4-8: Bulimia—Criteria for Hospitalization |
|---|
| Syncope |
| K < 3.2 |
| Cl < 88 |
| Esophageal tears |
| Cardiac arrhythmias, prolonged QTc |
| Hypothermia |
| Suicide risk |
| Intractable vomiting |
| Hematemesis |
| Failure to respond to outpatient management |

**Quick Quiz**

- True or false? Bulimic girls are usually underweight.
- What is the most likely type of breast mass found in an adolescent girl?
- When should you further investigate a breast mass?
- What is the most common cause of mastitis?

growth of the glandular ductal system. Obviously, other hormones, such as progesterone, play important roles. Progesterone affects alveolar growth, as do, among others, insulin and growth hormone. Estrogen requires the presence of these hormones for breast growth to occur.

Initial pubertal breast development occurs as a proliferation of ductal and stromal tissue and fat deposition, known as the "breast bud." Puberty results in both lobular-alveolar and ductal growth. Lactation occurs later and is due to the presence of prolactin and adrenal steroids.

Asymmetrical growth, where one breast develops more slowly than the other, is common; this may persist into adulthood. Breast atrophy is a sign of an eating disorder, with associated severe weight loss and loss of breast and fatty tissue. Accessory breast tissue (polymastia) and accessory nipples (polythelia) occur along the mammalian nipple line in 1–2% of girls. Juvenile hypertrophy, a massive enlargement of one or both breasts, may require mammoplasty after completion of breast maturation, particularly when the hypertrophy is severe.

Breast masses are common in the adolescent breast. Bloody discharge, skin dimpling, and nipple retraction are signs of concern for breast cancer but are extremely rare in adolescents. The most common breast masses are solitary cysts, fibrocystic changes, and fibroadenomas.

Most breast masses that do not regress or change with hormonal fluctuations turn out to be fibroadenomas, a benign mix of stromal elements, ducts, and acini. The mass is usually painless and rubbery in character. They are usually in the upper-outer quadrant of the breast and range from 1–3 cm. They have no malignancy potential.

A solitary cyst is the most common breast mass in the adolescent. > 50% of these cysts will resolve in 2–3 months and do not require further study; simply follow up with serial exams. You can use ultrasound if physical examination cannot differentiate cystic from solid masses. Asymptomatic solid breast masses that are < 5 cm and consistent with fibroadenoma can be observed. Persistent cystic lesions can be evaluated and/or treated with needle aspiration. Persistent larger or suspicious lesions should undergo excisional biopsy (wide margins are not usually indicated).

Recurrent cysts usually represent fibrocystic changes—known as "benign proliferative breast disease"—and are due to a physiologic response of breast tissue to cyclic hormones. The most common symptom of fibrocystic changes is bilateral pain in the upper-outer quadrants of the breast. The pain usually begins in the premenstrual cycle and stops soon after menses. Treat with support, using nonsteroidal medications for pain. Oral contraceptives also may reduce the frequency and duration of pain.

Do not use mammography for routine screening or for adolescents with breast masses, because the adolescent breast does not have enough fat in it to make a mammogram readable. Self-breast exam instruction is controversial, because the risk of breast cancer in this age group is near zero. There is the balance between increasing unnecessary anxiety (almost every mass identified is benign), and fostering a valuable, preventive-health routine in adolescents that can strengthen future behavior (self-exams) as an adult.

Cystosarcoma phyllodes, although rare, are rapidly growing lesions that have a small risk of becoming malignant. Intraductal papilloma is a benign, slow-growing tumor located under the areola. It may present with a serous, or sometimes bloody, discharge. Primary breast cancer is extremely rare in adolescents. Only 0.2–3% of all breast cancers occur in those younger than 25 years of age. Breast cancers present in adolescents as a hard, fixed mass with overlaying skin changes, frequently under the nipple. Family history is extremely important, but only ~ 6% will have breast cancer-associated cancer genes. Remember: ultrasound and biopsy are the procedures of choice.

Mastitis is a localized breast infection that is more commonly seen in newborns and lactating women. You might also see it occasionally in a nonpregnant adolescent. The most common organism identified is *Staphylococcus aureus*; thus, treat with antistaphylococcal antibiotics, heat, and analgesia.

### Amenorrhea

Primary amenorrhea refers to the absence of menses in one or more of the following:

1) By age 16 years with normal secondary sexual development (e.g., breast development)
2) By age 14 years in the absence of any breast maturation
3) By age 14 years in an individual with clinical stigmata of, or karyotype consistent with, Turner syndrome
4) Despite having attained sexual maturation rating 5 for ≥ 1 year or despite the onset of thelarche 4 years previously
5) By age 12–13 years if cyclic pelvic pain is present (Think about obstructed Müllerian outflow tract.)

Others more simply define primary amenorrhea as lack of menses by 16 years of age.

Secondary amenorrhea refers to loss of menses for > 3–6 consecutive months after previous regular cycles, or for > 9–12 months in those with previously irregular cycles. Note: Pregnancy is the most common reason for secondary amenorrhea and can also be an etiology of primary amenorrhea for some adolescents. Don't forget, though, that irregular cycles are common in adolescents, so the duration of amenorrhea may or may not be helpful in determining if true secondary amenorrhea is occurring. (Obviously, if an adolescent with irregular cycles presents with amenorrhea for 5 months, you are also going to consider pregnancy as an etiology.)

Causes of amenorrhea are central (hypothalamic or pituitary), ovarian, or anatomic (uterus, cervix, vagina).

Central: If the hypothalamus is the culprit, it is usually due to partial or complete inhibition of gonadotropin-releasing hormone (GnRH) release. This inhibition can be due to nutritional deficiencies, cystic fibrosis, anorexia nervosa, excessive exercise, stress, or a drug (e.g., phenothiazine-like products). If the inhibition of GnRH is due to eating disorders, exercise, or stress it is commonly called "functional hypothalamic amenorrhea." Local lesions in the hypothalamus that could disrupt release are comparatively very rare. Don't forget that isolated GnRH deficiency has been associated with Kallmann syndrome and the inability to smell (anosmia). Deficiency of pituitary gonadotropins (isolated gonadotropin deficiency is rare and pituitary gonadotropin deficiency is usually due to panhypopituitarism) may be the result of tumors (most commonly craniopharyngioma), infiltrative disease (e.g., sarcoidosis, hemochromatosis, tuberculosis), or infarction (Sheehan syndrome). Note: In women who are of reproductive age, the most common pituitary cause of amenorrhea is a prolactin-secreting adenoma (prolactinoma). Look for galactorrhea too, but it is found in only ~ 60% of young women with a prolactinoma—but almost always found on the ABP Board exam.

Ovarian: Remember that the ovary produces estrogen and progesterone; therefore, if the ovary is malfunctioning, menstrual irregularities will occur. Problems can include gonadal dysgenesis, premature ovarian failure, infection, hemorrhage, and autoimmune oophoritis. Note: 10% of primary amenorrhea is due to gonadal dysgenesis, frequently as a result of Turner (XO) syndrome. Secondary amenorrhea can also be associated with gonadal dysgenesis, particularly mosaic Turner syndrome!

Polycystic ovaries and chronic hyperandrogenic anovulation are common causes of secondary amenorrhea and, occasionally, primary amenorrhea. Although polycystic ovarian syndrome is a clinical diagnosis, a clue in 50% of patients with polycystic ovaries is an LH:FSH ratio > 2.5:1 and/or elevated levels of free testosterone, androstenedione, and dehydroepiandrosterone sulfate (DHEAS). This indicates a chronic hypothalamic dysfunction. Really, anything that increases circulating androgen hormones will result in amenorrhea, hirsutism, and virilization, as seen in polycystic ovaries. Examples include Cushing syndrome, anabolic steroid abuse, and adrenal adenomas.

Anatomic: This type of amenorrhea is frequently due to a defect in the development of the Müllerian duct system, resulting in imperforate hymen, vaginal atresia, or absence/malformation of the cervix or uterus. If any of these is present (with the exception of the absence of a uterus), normal menses occurs but is blocked, resulting in painful swelling above the blockage (hematocolpos). Rokitansky syndrome is Müllerian agenesis with primary amenorrhea and absence/hypoplasia of the vagina, cervix, and/or uterus. Asherman syndrome occurs with uterine synechiae occurring after endometrial disruption/infection, leading to secondary amenorrhea with obstruction/obliteration of the uterine cavity.

How do you assess amenorrhea? Evaluate with the usual complete history and physical but, of course, pay particular attention to where in the hypothalamus-pituitary-gonadal axis there might be a problem. Premature or excess pubic, axillary, and facial hair development indicate androgen defect/problems. Look at breast and vaginal mucosa for maturation effects of estrogen.

Always perform a pregnancy test first! Then ask about things that could cause "central" etiologies, such as weight loss, nutrition, excessive exercise, and/or stress. Look for any physical signs of Turner syndrome and for galactorrhea (prolactinoma).

Signs of androgen excess: hirsutism, acne, voice changes, and clitoromegaly. Pelvic examination is important to identify possible anatomic etiologies.

Lab screens after a negative pregnancy test usually include: LH and FSH to differentiate between a hypothalamic and an ovarian etiology; prolactin level to determine a pituitary microadenoma; and TSH for thyroid difficulties. If you suspect androgen excess during the physical exam, order free testosterone and dehydroepiandrosterone sulfate levels.

While waiting on these results, perform a progesterone challenge to evaluate if estrogen is present and the anatomy is normal. Give 10 mg of medroxyprogesterone for 7 days, and expect bleeding by days 2–7 after completion of the course. If no bleeding occurs, the anatomy is disrupted or there is not enough estrogen. Next, give 2.5 mg oral conjugated estrogen (Premarin®) for 25 days with 10 mg oral medroxyprogesterone added from day 16–25. If still no bleeding occurs, order pelvic sonogram/CT and hormonal levels, including estradiol levels. Anatomic abnormalities resulting in obstruction usually require surgical correction.

**QuickQuiz**

- What is the most common reason for secondary amenorrhea?
- Amenorrhea and anosmia should make you consider what syndrome?
- What is the most likely etiology of galactorrhea and amenorrhea in a female adolescent?
- What is significant about the LH:FSH ratio in girls with polycystic ovary disease?
- Describe signs of androgen excess in amenorrhea.
- A girl presents with her first menses and hemorrhages to a hematocrit of 20%. It is difficult to stop her bleeding. What is the likely etiology for her excessive blood loss?
- What type of cancer is seen in daughters of mothers who took diethylstilbestrol (DES)?

Another method begins with whether or not a uterus is present and then following the algorithm in Figure 4-1.

Finally, polycystic ovaries frequently require cycling with estrogen and progesterone (usually combination oral contraceptives that contain low androgenic progestins, such as desogestrel and drospirenone). Hirsutism may require electrolysis and treatment with spironolactone—an antiandrogenic agent that works by competing at the androgen receptor site and by inhibiting 5-alpha-reductase, thus reducing conversion of testosterone to dihydrotestosterone. If you find a pituitary microadenoma, use bromocriptine. True ovarian failure requires hormone replacement therapy with larger doses of conjugated estrogens and medroxyprogesterone on days 16–25.

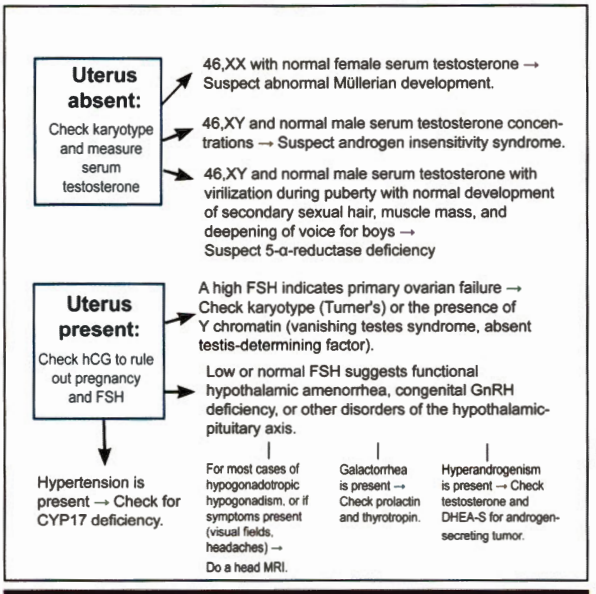



**Figure 4-1:** Algorithm to Assess Amenorrhea – Uterus Present or Not

### Dysfunctional Uterine Bleeding

Dysfunctional uterine bleeding is defined as vaginal bleeding that occurs in cycles < 20 days or > 45 days apart, lasts > 8 days, results in blood loss > 80 mL, and/or is associated with anemia. Dysfunctional uterine bleeding is further subdivided into primary (most common) and secondary.

Primary dysfunctional uterine bleeding is usually due to an immature hypothalamus-pituitary-gonadal system. Anovulatory cycles are common early in most adolescents and are characterized by large variations in estrogen levels and a lack of progesterone. During the first 2 years following menarche, anovulation is associated with 50–80% of bleeding episodes, falling to 30–55% 2 to 4 years after menarche and to 20% 4 to 5 years following menarche.

Treatment of anovulatory dysfunctional bleeding depends on the severity of bleeding, hemoglobin level, and the degree, if any, of associated hemodynamic changes. Mild cases associated with a hemoglobin level of ≥ 12 g/dL require only reassurance, a multivitamin with iron, and close follow-up. Individuals with a hemoglobin level of 10–12 g/dL should take a 35 μg combined oral contraceptive every 6–12 hours for 24–48 hours until the bleeding stops, taper to one pill per day by day 5, and then begin a 28-day combined oral contraceptive packet. This regimen should be continued for 3–6 months. Patients with a hemoglobin level of < 10 g/dL may require hospitalization and initial treatment with higher doses of estrogen if hemodynamically unstable. All women with dysfunctional bleeding will need iron supplementation.

Secondary dysfunctional bleeding is usually due to coagulation disorders and underlying disease processes of the vagina, cervix, uterus, and ovary. The most common cause of excessive bleeding that requires hospitalization is the presence of a bleeding disorder. Abnormal bleeding at the time of menarche may be the first sign for some of these diathetic bleeding disorders, especially von Willebrand disease. Other causes to consider include Factor VIII or IX deficiency, thrombocytopenia, platelet disorders, thalassemia major, and leukemia.

Anatomic causes include lacerations to the vagina, hymenal tears, and foreign bodies. Diethylstilbestrol (DES), used in the 1960s and early 1970s, can cause a rare, DES-related clear cell adenocarcinoma in daughters of mothers who took it, which may present as vaginal bleeding. Cervical problems, such as STDs and polyps, also can cause bleeding. Endometritis is an etiology of uterine origin and usually results from a preceding infection.

Treatment of secondary dysfunctional bleeding requires attending to the primary problem, which usually

involves assistance from a gynecologist, and will not be discussed further here. However, if you discover an underlying cause prior to such referral, obviously you should address it.

### Dysmenorrhea

Dysmenorrhea is one of the leading causes of school absenteeism in adolescents, because over 2/3 of teenage girls have it. We have a pretty good idea what causes dysmenorrhea. Most dysmenorrhea is due to prostaglandin production before menses, which causes vasoconstriction and muscular contractions. Remember: The cyclooxygenase pathway of arachidonic acid makes $PGE_2$, $PGD_2$, and $PGF_{2\alpha}$, which cause vasoconstriction, followed by vasodilation, and then muscle contraction and relaxation. The biggest increase in the prostaglandins occurs just before menses. Alternatively, leukotrienes are involved in some women, because they are produced during the last steps of the lipoxygenase pathway. These agents stimulate smooth muscle to contract.

So, based on this information, give cyclooxygenase inhibitors like ibuprofen or naproxen for dysmenorrhea in hopes that it is due to the more common cyclooxygenase pathway. If it commonly occurs and is predictable during the cycle, it may be best to start these inhibitors before the symptoms begin. For women who don't respond, their pain is likely due to the lipoxygenase pathway. Besides agents that inhibit the synthesis of prostaglandins (ibuprofen, naproxen), oral contraceptives are very effective in reducing or eliminating dysmenorrhea.

Secondary dysmenorrhea is due to other conditions, such as infection, ectopic pregnancy, endometriosis, anatomical abnormalities, ovarian cyst or mass, and other etiologies.

### Premenstrual Syndrome (PMS) and Premenstrual Dysphoric Disorder (PMDD)

PMS and PMDD present with physical and behavioral symptoms that occur in the second half of the menstrual cycle, especially during the first few days of menses. The most common findings are abdominal bloating, fatigue, breast tenderness, and headaches. To meet criteria for PMDD as defined in the DSM-IV, PMS and its somatic findings (bloating, breast tenderness, etc.) occur with the presence of at least one affective disorder, such as depression, anger, irritability, confusion, social withdrawal, or fatigue. PMS occurs in up to 30% of women and PMDD in 3–8%.

Various therapeutic agents are useful. Serotonin reuptake inhibitors (fluoxetine, sertraline, paroxetine, and citalopram) have been shown to be effective, as have oral contraceptives. Alprazolam, GnRH agonists, and danazol have been used. There is also some evidence that exercise, relaxation techniques, and reflexology may alleviate PMS symptoms. Finally, limited studies have shown benefits from calcium, vitamin $B_6$ and vitamin E supplements, and the herbal preparation *Agnus castus*.

## Male Issues

### Breast—Gynecomastia

Gynecomastia occurs in ~ 50% (studies range though from 4–69%!) of boys ages 10–16 years and is most commonly seen around age 14. It is due to a decreased ratio of androgen to estrogen and a change in the sensitivity of breast tissue receptors. The area of enlargement is usually tender and asymmetric. Most gynecomastia resolves spontaneously within 3 years. Rare causes of gynecomastia to look for (especially on the Boards) are Klinefelter syndrome; anabolic steroid abuse; tumors of the testicles, adrenal, or pituitary; medications (psychoactive drugs, digoxin, anti-hypertensives, ketoconazole, cimetidine, and phenytoin); and some illicit drugs, such as marijuana and amphetamines. "Normal" pubertal gynecomastia is usually < 4 cm and does not require specific workup or therapy because most will resolve spontaneously. If it is "macrogynecomastia" and > 5 cm (similar to a 2–3 Tanner female), it usually will not regress and may require surgical therapy; recent studies show promise using tamoxifen and testolactone.

### Scrotal Masses

3 types of scrotal masses may occur during adolescence: congenital, acquired, and infectious.

1) Congenital masses include scrotal dermoid, polyorchism, and scrotal rests (abnormal tissue from other organ systems, such as spleen or adrenals). Congenital masses usually require surgery.
2) Acquired lesions include hydroceles, spermatoceles, varicoceles, torsion of the spermatic cord or the testicular appendage, trauma/hematocele, and neoplasms. Each of these is described in more detail below.

   Hydroceles are nontender and fluid-filled masses that collect between the parietal and visceral layers of the tunica vaginalis. A new hydrocele may be associated with a hernia or testicular lesion and should be investigated. Evaluate with ultrasound. Also, they can be transilluminated. Generally, no treatment is required unless they become painful or too large.

   Spermatoceles (retention cyst of the epididymis containing spermatozoa) are located in the efferent ductal system and will present as a nodule above and posterior to the testes. Key things to remember here are: There is no change with Valsalva, and they transilluminate. These do not affect fertility and generally do not require therapy.

   Varicoceles are dilated scrotal veins and are usually idiopathic but may be secondary to intraabdominal masses, hepatosplenomegaly, and other disorders. In the physical examination on a Board exam question, you will see the words "bag of worms" as a descriptor. Varicoceles are usually left-sided and increase

**QuickQuiz**

- What is the usual etiology for dysmenorrhea?
- True or false? Gynecomastia in boys is rare.
- True or false? Gynecomastia in boys is usually tender and asymmetric.
- Define hydroceles, spermatoceles, and varicoceles.
- True or false? Torsion of the spermatic cord is a surgical emergency.
- What is the most common cause of epididymitis in an adolescent male?

with Valsalva. They can decrease spermatogenesis if they last a long time, and you will need to perform surgery if the testicle has become hypotrophic on that side; therefore, remember that loss of testicular volume (> 2 mL difference in testicular volume) or failure of the testes to grow during puberty are indications for surgery.

Torsion of the spermatic cord is a surgical emergency! The patient presents with acute onset of severe pain and swelling in the testis, inguinal area, and possibly lower abdomen—usually with accompanying nausea/vomiting. The exam will show a diffusely swollen and tender testicle and an absent cremasteric reflex. A radionucleotide scan will show decreased uptake in the scrotal images. You might also consider a Doppler ultrasound. A urologist may attempt a manual "detorsion"; if this is not successful, the patient requires emergency surgery for either an orchiopexy or orchiectomy (if necrotic).

Torsion of the testicular appendage affects the appendix testis or appendix epididymis. A common symptom is either sudden or gradual pain at the upper pole of the testis. The examination will show a tender, pea-sized swelling at the upper pole of the testis and the "blue dot" sign (a blue hue visible through the scrotum). Doppler flows and radio-nuclide scans will show normal-to-increased values (as compared to torsion of the spermatic cord). Treat with analgesics and antiinflammatories; the torsion will resolve spontaneously in 2–12 days without surgery.

Trauma/hematocele is, as the name implies, due to trauma. It is associated with onset of pain from the trauma, and swelling may or may not be present. Use ultrasound if you are concerned about an enlarging hematocele. Analgesia, ice, and scrotal elevation are helpful. If the hematocele continues to enlarge, surgical drainage is likely necessary.

Testicular neoplasms usually present as painless swelling—unless hemorrhage or necrosis has occurred, which then typically causes pain. The patient may complain of back pain if retroperitoneal lymph nodes are present. 95% are germ cell tumors (most commonly seminoma, embryonal carcinoma, teratoma, and choriocarcinoma), and the other 5% are of stromal tissue origin. Physical exam will usually show an irregularly shaped, firm mass within the testis that does not transilluminate. Workup includes hCG (increased in choriocarcinoma and mixed germ cell tumors), α-fetoprotein (yolk-sac tumors and embryonal carcinoma), testicular ultrasound, and CT scan of chest and abdomen. Note: Most seminomas do not produce any markers! Scrotal masses are evaluated with ultrasound or MRI. Treatment includes orchiectomy and peritoneal lymph node dissection, as well as radiation and chemotherapy, depending on the staging. Pretreatment sperm banking is recommended. Overall survival is 70% and > 95% in stages I and II.

3) Infectious etiologies of scrotal masses include epididymitis and orchitis. In adolescents, epididymitis is usually due to an STD, and in decreasing frequency, to *C. trachomatis*, *N. gonorrhoeae*, *E. coli*, *Pseudomonas*, or gram-positive cocci. In men who have sex with men, unprotected anal intercourse may cause epididymitis due to *E. coli* and other bowel flora. Pain is usually acute at onset. Patients will present with increased urinary frequency, dysuria, urethral discharge, and fever. The examination will show a swollen and tender epididymis. In contrast to torsion, pain due to epididymitis is often relieved by elevation of the testis (Prehn sign). U/A, Gram stain, and cultures of discharges and urethra are usually diagnostic. To differentiate epididymitis from torsion, radionucleotide scan and Doppler will show increased uptake and flow, respectively, compared to torsion. Azoospermia can result, even with appropriate therapy. Therapy is discussed later in this section, and the key is to treat both *Chlamydia* and gonorrhea.

Orchitis is usually a result of preceding epididymitis. Here, the patient presents with a swollen and tender testicle and fever. If mumps is involved, a preceding or concurrent parotitis is likely. Workup and treatment of orchitis are the same as with epididymitis—unless mumps is likely, in which case just supportive care is indicated. If atrophy occurs after orchitis, there is a high risk of cancer. Bilateral atrophy usually leads to infertility.

## CONTRACEPTION

Unfortunately, contraception discussions with adolescents come up, on average, 6+ months after initial sexual intercourse. With women, these discussions frequently occur during a visit for a missed period. There are numerous forms of contraception available, but the only one that has 100% efficacy is abstinence. However, some other contraceptives get pretty close to that surety

level. Oral contraceptives, for example, are 99.9% effective if used correctly. IUDs are 98–99% effective. Male condoms are 97% effective if used "perfectly," but, in real-world situations, are about 87% effective. Female condoms are 95% effective if used as directed by the manufacturer but, in real-world settings, are only 79% effective. A diaphragm with spermicide is 94% effective if used as directed, but in the real world, the rate is ~ 80%. Injectables, such as depo-medroxyprogesterone, are 99% effective in real-world settings. "Periodic abstinence" or "rhythm" methods result in 90–94% effectiveness in "perfect" usage, but in real-world settings, the effectiveness is ~ 80%. The only method, besides abstinence, that prevents STDs is male condoms; recommend them to adolescents, even if they are using other forms of birth control, to reduce the risk of sexually transmitted diseases.

Absolute and relative contraindications to oral contraceptives (OCPs) are listed in Table 4-9.

Emergency contraception became a topic of increasing conversation in the United States with the release of mifepristone (RU-486) in 2000. This agent prevents 100% of pregnancies if taken within 72 hours of intercourse. Levonorgestrel (Plan B®) is approved for over-the-counter sales to women > 17 years of age and by prescription only for those < 17 years of age. This agent is most beneficial in preventing pregnancy if used as soon as possible, but also up to 120 hours after unprotected intercourse. Other methods include combination pill formulations that contain 100 µg ethinyl estradiol + 0.5–1 mg norgestrel, with a second pill taken 12 hours later. These agents will work 74% of the time if used in the first 24 hours, but have significant side effects, including nausea/vomiting and menstrual irregularities. Numerous other combinations are reported in the literature. Because of its ease of use and better safety profile, levonorgestrel has become the drug of choice.

**Table 4-9:** Contraindications for Oral Contraceptives

| |
|---|
| **Absolute** |
| Abnormal vaginal bleeding of unknown cause |
| Estrogen-dependent tumor |
| Liver disease |
| Thromboembolic disease |
| Cerebrovascular events |
| Migraine with aura |
| **Relative** |
| Diabetes mellitus |
| Seizures |
| Vascular headaches (includes migraines **without aura**) |
| Severe/moderate hypertension |
| Tobacco use |

# SEXUALLY TRANSMITTED DISEASES

## OVERVIEW

### Prevalence of STDs

Well, you knew this would have to be in the Adolescent Health section, right? > 50% of adolescents have had sex by age 16, and 75% by age 19. The average age for first-time sexual encounter is 16 years, but is much lower in inner city youth. Adolescents commonly become infected with sexually transmitted diseases. Prevalence rates vary, but 10–25% of sexually active adolescents have *Chlamydia trachomatis* (many asymptomatic). The reported rates of *Chlamydia* and gonorrhea are highest among females 15–19 years of age. Positivity rates for herpes simplex virus (HSV) are as high as 30% in some adolescent populations; most are asymptomatic. Of additional concern is that 20–60% of sexually active female adolescents have human papillomavirus DNA, which can predispose to cervical cancer. This section will cover each of the STDs, hitting on the important things you need to know for each. But first, some closely related topics.

### Prevention

Abstinence is the most reliable way to prevent STDs. However, we are dealing with adolescents (see above % who are sexually active), so a male condom is the cheapest and—thinking "real world" here—the most effective means of prevention available. Female condoms appear to be effective as mechanical barriers for viruses, including HIV; however, no studies have really evaluated the effectiveness of these in the prevention of STDs. Vaginal spermicides are not effective in preventing STDs, and rectal spermicides actually may increase risk of transmission because of their role in facilitating damage to rectal cell linings. Vaginal sponges and diaphragms are protective against cervical gonorrhea and *Chlamydia;* however, teens should never rely on either of them to prevent HIV. Don't forget that hepatitis B is a sexually transmitted disease, and preexposure vaccination is very effective in preventing transmission! Recommend the hepatitis A vaccine to men who have sex with men (MSM) and/or with illegal drug users (both injection and non-injection). But also remember that the hepatitis A and HPV vaccines are now recommended for all children.

### Reporting and Confidentiality

Syphilis, gonorrhea, *Chlamydia*, and AIDS are reportable diseases in every state. HIV infection and chancroid are reportable in many states. For reporting other STDs, the requirements vary by state. The laboratory or the provider, depending upon the state, can do reporting. STD reports are strictly confidential. In most states, they are, by statute, protected from subpoena.

**Quick Quiz**

- Which form of contraceptive is also effective for preventing sexually transmitted diseases?
- Name the absolute contraindications for using oral contraceptive pills.
- How common is sex among adolescents?
- Who has the highest prevalence rates of gonorrhea—males or females?
- What are the "reportable" diseases in every state?
- What screening laboratory tests should be done or offered to a pregnant adolescent?
- For which viral infection is prophylactic cesarean delivery indicated? Under what conditions?
- Describe the acute HIV retroviral syndrome.

### Pregnant Adolescents

Current CDC guidelines recommend that all pregnant women be tested for HIV. Also do all of the following at the first prenatal visit: Syphilis testing, hepatitis B surface antigen (HBsAg), testing for *Chlamydia trachomatis* and *N. gonorrhoeae*, hepatitis C antibody testing, and rubella titers, as well as Pap smears at the same frequency as nonpregnant women.

Do not perform routine serial cultures for herpes simplex virus (HSV) during pregnancy. You must perform prophylactic cesarean section only if active lesions are present at the time of labor/delivery. The presence of genital warts is not an indication for cesarean section. (Note that the idea of monitoring pregnant women for HSV is evolving. According to the 2009 Red Book, there is growing evidence that type-specific antibody avidity testing may prove useful for evaluating risk of neonatal infection. The presence of low-avidity HSV-2 IgG in serum of near-term pregnant women has been correlated with an elevated risk of neonatal infection—however, no current guidelines specifically recommend doing cultures or antibody testing in pregnant women.)

Do not conduct routine screening of pregnant women for *Trichomonas vaginalis*.

### Children with STDs

Children (i.e., not consenting adolescents) who have STDs are, by definition, victims of sexual abuse. Treatment requires incorporating the use of highly skilled individuals, including the pediatrician, laboratory personnel, social workers, psychologists, and child protection authorities. Gonorrhea, syphilis, and *Chlamydia*, if acquired after the neonatal period, are 99.99% indicative of sexual contact. For other diseases, such as human papillomavirus and vaginitis, the association is not as clear.

### HIV

Guidelines for screening for HIV infection have changed markedly since 2006. Now the CDC recommends screening all patients aged 13–64 years of age at routine visits, unless you can document in your practice that the HIV prevalence is less than 0.1% (< 1 per 1,000 patients screened). Also, screening is recommended as "opt-out," meaning patients should be informed orally or in writing that HIV testing will be performed unless they specifically decline. A separate HIV consent form is no longer recommended or required. This is markedly different from previous screening guidelines that required written, "informed" consent. Obviously, these guidelines have caused quite a controversy. But remember: For Board purposes, follow the guidelines. For sure, recommend testing for HIV for all who seek evaluation and treatment for STDs, as well as for those exhibiting high-risk behaviors who present with other conditions. Counseling before and after testing is no longer an integral part of the "screening" procedure but should remain integral in the setting of high-risk behaviors or at STD clinics.

HIV infection is diagnosed by testing for antibodies against HIV-1 and HIV-2. First, you order antibody testing with a sensitive screening test—the EIA (enzyme immunoassay; formerly ELISA). A Western blot (WB) or an immunofluorescence assay (IFA) confirms repeatedly reactive EIA tests. If the confirmatory test is positive, the patient has HIV infection and is presumed infectious to others. Within 3 months of infection, 95% of those infected will have positive antibodies. More than 99% of infections in the U.S. are due to HIV-1. A positive "rapid" oral/serum/plasma HIV test must be confirmed with a conventional blood HIV test.

Look for the acute retroviral syndrome (especially on the Board exam). It is characterized by fever, malaise, lymphadenopathy, and skin rash. (Granted, it looks and sounds like any viral infection, but be suspicious if the Boards present an adolescent with specific risk factors for HIV in the patient's history!) The syndrome usually occurs in the first few weeks of infection, before antibody testing is confirmed positive. The test to order for the acute retroviral syndrome is an HIV PCR-DNA test or, in some centers, HIV plasma RNA (viral load) is used. Current guidelines report possible benefit from early therapy in these patients; quickly refer to an HIV specialist to help make this determination. Further treatment/evaluation of HIV-infected patients is covered in the Infectious Disease section.

## STDs CHARACTERIZED BY GENITAL ULCERS

### Overview

In the U.S., there are 3 possibilities for STDs characterized by genital ulcers: herpes, syphilis, or chancroid. Granuloma inguinale (Donovanosis) occurs more prominently in other parts of the world.

The 2010 CDC STD guidelines say that diagnosis of genital ulcers on history and physical is inaccurate and that any patient with a genital ulcer should have specific tests for evaluation of genital, anal, or perianal ulcers including:

- Culture for HSV or PCR testing for HSV
- Serologic testing for type-specific HSV antibody
- Syphilis serology and dark-field examination

## Herpes Simplex

Genital herpes is a recurrent, lifelong infection. There are 2 types of HSV: HSV-1 and HSV-2. Most cases of genital herpes are HSV-2. > 50 million people in the U.S. have genital herpes infection. Most of these patients have mild or unrecognized infection but still shed the virus intermittently! Typical lesions are painful, itchy, and with multiple vesicles (remember "tender grouped vesicles"—a clue that helps to differentiate genital herpes from syphilis and chancroid) (Image 4-1). Vesicles spontaneously rupture to form shallow painful ulcers. The average duration of initial infection is 12 days; recurrent infections have an average duration of 4–5 days.

Primary HSV is commonly asymptomatic but the Boards tend to focus on the symptomatic patients. (I guess it is hard to ask a question about an "asymptomatic" person …) Look for low-grade fever, inguinal lymphadenopathy, vesicular lesions, cervical motion tenderness, and a thin, white vaginal discharge. But don't be swayed by the presence of cervicitis and discharge and think it is something else (obviously they can be coinfected with gonorrhea or *Chlamydia*), but if they say "vesicles" think HSV!

Diagnosis: Isolation of HSV in cell culture is the preferred virologic test in patients who present with genital ulcers, especially since the typically painful, multiple vesicular lesions or ulcerative lesions are absent in many infected individuals.

Reliable, type-specific serologic tests are available. Several glycoprotein G (gG)-based, type-specific assays have been approved by the FDA, with sensitivities and specificities of 90–100%. Use these serologic tests in those who have presumed false-negative HSV cultures to diagnose those with unrecognized infection and to manage sex partners with genital herpes.

Courtesy of CDC N.J. Fiumara, MD, Gavin Hart, MD

*Image 4-1: HSV-2*

PCR testing is not FDA-approved for genital specimens but is the test of choice for identifying HSV infection of the CNS.

Tzanck preparations are insensitive, nonspecific, and pretty much appear only on Board exams now! (See Image 4-2, showing a positive Tzanck prep with the classic multinucleated giant cells.)

Treatment of genital herpes: Offer antiviral therapy since it is beneficial to most patients. Newly acquired genital herpes can cause a prolonged clinical illness with severe genital ulcerations and neurologic involvement. These drugs will partially control symptoms in initial episodes or recurrences and can be used to suppress future outbreaks. They are most helpful if initiated within 24 hours of symptoms. For episodic, recurrent therapy, many patients experience a prodrome of itching and burning. Initiate therapy before physical appearance of lesions; however, these drugs do not eradicate latent virus nor do they modify recurrences once they are stopped. Three drugs are approved: acyclovir, valacyclovir (the valine ester of acyclovir provides better oral absorption), and famciclovir (a prodrug of penciclovir, with better oral absorption than acyclovir). Topical therapy is worthless.

Note: Don't worry about memorizing specific dosages of drugs listed for herpes and other STDs, unless specifically told they are important. Whew!

First clinical episode of genital herpes:

- Acyclovir 400 mg tid or 200 mg 5/day x 7–10 days, or
- Famciclovir 250 mg tid x 7–10 days, or
- Valacyclovir 1 g bid x 7–10 days

Episodic therapy for recurrent genital herpes:

- Acyclovir 400 mg tid or 800 mg bid x 5 days, or
- Acyclovir 800 mg tid x 2 days, or
- Famciclovir 125 mg bid x 5 days or 1,000 mg bid x 1 day, or
- Valacyclovir 500 mg bid or 1g/day x 3–5 days

Suppressive therapy for recurrent genital herpes:

- Acyclovir 400 mg bid daily, or
- Famciclovir 250 mg bid daily, or
- Valacyclovir 500 mg or 1 gm q day

[Know]: Even with suppressive and other therapies, subclinical viral shedding still occurs, so encourage adolescents to continue to use condoms. Suppressive therapy reduces the frequency of genital herpes recurrences by 70–80% in patients who have frequent recurrences.

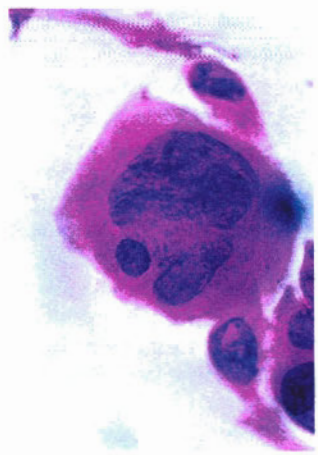


*Image 4-2: Giant Cells (Tzanck smear)*

**Quick Quiz**

- Differentiate the genital lesions of HSV from syphilis.
- What defines a positive Tzanck prep?
- True or false? Patients on herpes-suppressive therapy who do not have active lesions still may be shedding virus.
- Describe the palmar rash of secondary syphilis.

For those with severe disease or those with disseminated infection, use IV acyclovir 5–10 mg/kg IV q 8 hours.

Herpes and pregnancy: The risk of transmission to the neonate from an infected mother is very high: 25–60% if the mother has her first episode of genital herpes near delivery. The risk of transmission is ~ 2% if she acquired the infection in the first half of pregnancy, or if she has recurrent herpes at term. > 75% of infants who contract HSV are born to women who had no history of HSV infection (asymptomatic).

Women who have no signs or symptoms (or prodrome) of herpes at delivery may deliver vaginally. Perform cesarean section if lesions are present at the time of delivery. Do **not** take routine viral cultures at the time of delivery in an asymptomatic patient since these do **not** predict viral shedding. Neonatal herpes infection is associated with high mortality rates; many survivors have severe ocular and neurological sequelae.

## Syphilis

### Overview

Syphilis is a systemic illness due to the spirochete *Treponema pallidum*. There are 3 well-described stages of syphilis, and patients may present at any stage for treatment.

**Primary** infection is characterized by a painless ulcer or chancre at the site of infection, often unrecognized and occurs ~ 3 weeks after exposure. The ulcer is typically described as a "punched out," clean-appearing ulcer with sharp, firm, slightly elevated borders (Image 4-3). Lesions are associated with firm, nontender bilateral regional lymphadenopathy.

**Secondary** infection generally occurs 1–2 months later and can vary in appearance and presentation but frequently will present with a scaly, hyperkeratotic palmar skin rash, lesions on the trunk and extremities that tend to follow the lines of cleavage, mucocutaneous lesions, and lymphadenopathy. Additionally, splenomegaly, fever, malaise, and joint pain can occur. Hypertrophic granulomatous lesions (condylomata lata) may occur in warm, moist areas, usually the vulva or anus. Classically, the rash is described as "nickel and dime" lesions on the palms and soles and will resolve spontaneously without treatment in 3–12 weeks (Image 4-4). For Boards, remember that a pityriasis-like rash that follows the lines of cleavage on the trunk and also involves the palms and soles is secondary syphilis. Generalized lymphadenopathy, fever, malaise headache, splenomegaly, and sore throat are common during secondary infection.

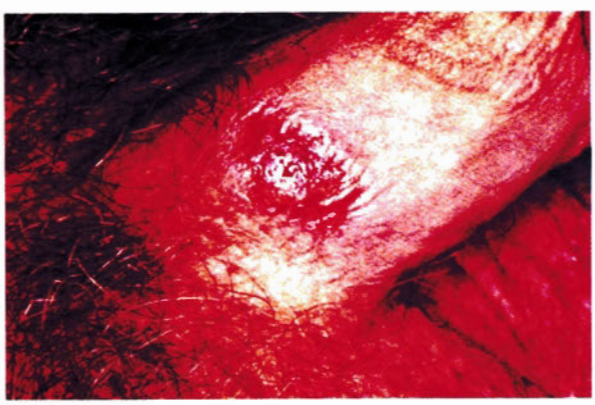
**Tertiary** infection presents 15–30 years later with cardiac, ophthalmic, and auditory abnormalities, as well as gummatous lesions of the skin, bone, or viscera.

Latent infections, those without clinical manifestations, are detected by serologic testing. Latent syphilis acquired during the preceding year is known as "early latent syphilis." The patient is generally infectious. "Late-latent syphilis" generally occurs after the first year of infection.

### Diagnosis

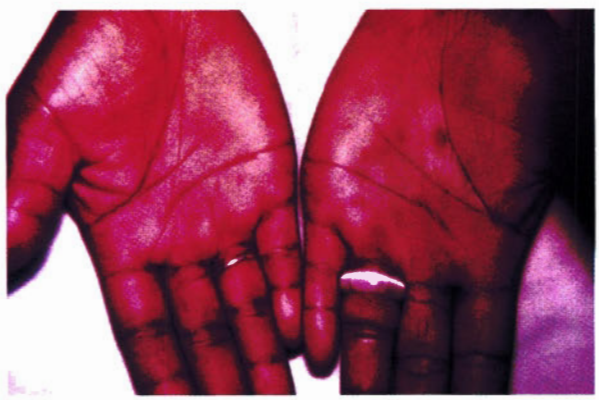
If lesions are present, dark-field examination or direct fluorescent antibody testing of the exudate or tissue is diagnostic. A presumptive diagnosis is possible using 2 serologic tests for syphilis in sequence:

1) **Non**treponemal tests (Venereal Disease Research Laboratory [**VDRL**] and Rapid Plasma Reagin [**RPR**])
2) Treponemal tests (fluorescent treponemal antibody absorbed [**FTA-ABS**] and *T. pallidum* particle agglutination [**TP-PA**])



Courtesy of CDC: Gavin Hart, MD, N.J. Humara, MD

*Image 4-3: Primary Syphilis, Painless Chancre*



Courtesy of CDC

*Image 4-4: Secondary Syphilis, "Nickel and Dime" Rash*

So first get the nontreponemal test, and then the confirmatory treponemal test. Which one you use doesn't really matter. It mainly depends on the laboratory you are using. The nontreponemal tests correlate fairly well with disease activity. A 4-fold rise or fall in titer is considered a clinically significant difference. Note: You cannot compare a VDRL with an RPR. You must use the same test. Nontreponemal tests usually become nonreactive with time after treatment. However, some patients can persist with low titers; this is known as a "serofast" reaction.

On the other hand, for most patients, treponemal tests (e.g., FTA-ABS) will be positive for life, regardless of treatment. About 15–25% who are treated for primary syphilis will serorevert after 2–3 years. Because of this, the treponemal tests are not good for looking at therapeutic responses!

Remember all patients with syphilis should be screened for HIV!

Neurosyphilis can be difficult to diagnose in a patient with a positive serum test. If the CSF-VDRL is positive, the patient has neurosyphilis. It is a very specific test. In contrast, a negative CSF-VDRL is very insensitive and will miss many people who really have neurosyphilis. Usually, the diagnosis of neurosyphilis relies on a variety of clinical and laboratory findings: elevated CSF WBC count and elevated CSF protein, with or without clinical manifestations. Recently, some have advocated getting an FTA-ABS test on CSF. It is less specific than a CSF-VDRL, but it is very, very sensitive. Some believe that a negative CSF FTA-ABS excludes neurosyphilis, but this point is still controversial.

### Treatment: Drug of Choice for Syphilis

Parenteral penicillin G is the drug of choice for all stages of syphilis. It is the only drug to use in a pregnant woman. If she is penicillin-allergic, desensitize her! The Jarisch-Herxheimer (allergic) reaction is an acute febrile response accompanied by headache, myalgia, and other symptoms within the first 24 hours after beginning treatment for syphilis.

### Treatment of Primary, Secondary, and Early Latent Syphilis

Benzathine penicillin G 2.4 million units IM x 1 dose (if the patient is < 48 kg, dose with 50,000 units/kg IM)

or

Doxycycline 100 mg bid x 14 days or tetracycline 500 mg qid x 14 days (use only if non-pregnant and penicillin-allergic!)

Patients should be reexamined clinically and serologically (VDRL or RPR) at 6 and 12 months following treatment. Assume patients have failed therapy or been reinfected if they have signs or symptoms that persist, or recur, or if they have a sustained 4-fold increase in titer. Treatment at this point is usually weekly injections of the benzathine penicillin G 2.4 million units IM for 3 weeks. Also evaluate these patients for HIV, and perform a CSF analysis.

### Treatment of Late-Latent Syphilis, Syphilis of Unknown Duration, or Tertiary Syphilis

Benzathine penicillin G 2.4 million units IM q week x 3 weeks (if the patient is < 48 kg, use a dose of 50,000 units/kg IM)

or

Doxycycline 100 mg bid x 28 days or tetracycline 500 mg qid x 28 days (only for non-pregnant, penicillin-allergic)

Evaluate patients with latent syphilis or syphilis of unknown duration for evidence of tertiary disease (aortitis, gumma, iritis). If the patient has neurologic or eye signs/symptoms, evidence of active tertiary syphilis (aortitis, gumma, iritis), treatment failure, or HIV infection, then also perform a CSF evaluation. Treatment for tertiary syphilis without CNS disease is the 3-week regimen listed above.

Follow-up includes repeat testing at 6, 12, and 24 months.

### Treatment of Neurosyphilis

Know that CNS disease can occur at any stage of syphilis, from primary to tertiary. Clinical clues of neurologic involvement include: cognitive dysfunction, motor or sensory deficits, ophthalmic or auditory symptoms, cranial nerve palsies, or classic meningitis symptoms. Note: A classic Board presentation is an adolescent with hearing loss (8$^{th}$ nerve deafness)! In this scenario, always think neurosyphilis! CSF is indicated in all of these cases. Also, don't forget about uveitis!

Preferred: Aqueous crystalline penicillin G 3–4 million units IV q 4 hours, or 18–24 million units/day as continuous infusion for 10–14 days.

Alternate: Procaine penicillin 2.4 million units IM q day + probenecid 500 mg qid x 10–14 days.

(Some experts will also give benzathine penicillin G 2.4 million units IM q week x 3 weeks at the end of neurosyphilis therapy.)

If penicillin-allergic, you can use ceftriaxone 2 grams/day IM/IV x 10–14 days (but some get cross-reactivity); most desensitize the patient instead.

Follow-up: If you initially find increased CSF WBCs, repeat CSF every 6 months until CSF WBCs are normal. CSF-VDRL and CSF protein decrease much more slowly than CSF WBCs and may not be normal until ~ 2 years out. If the CSF is not normal after 2 years, retreatment is probably indicated.

### Congenital Syphilis

Diagnosis of congenital syphilis is complicated because we have no definitive test for the infant. Transplacental

**Quick Quiz**

- How do you diagnose syphilis?
- A pregnant woman is diagnosed with primary syphilis. She has a history of anaphylaxis to penicillin. What is the best treatment for her?
- What are the specific treatments for primary syphilis, secondary syphilis, and late-latent syphilis?
- Describe how to diagnose and treat neurosyphilis.
- An infant is diagnosed with congenital syphilis. What are possible physical findings to look for? What is the specific treatment necessary?

transfer of IgG antibodies occurs, as does transfer of maternal nontreponemal and treponemal antibodies.

If an infant is born to a mother with a +VDRL or +RPR, examine the infant for clinical findings of congenital syphilis:

- Lymphadenopathy
- Nonimmune hydrops
- Jaundice
- Hepatosplenomegaly
- Hemolytic anemia and thrombocytopenia
- Pneumonia alba
- Nephrotic syndrome
- Rhinitis ("snuffles")
- Pseudoparalysis of an extremity secondary to osteochondritis (Image 4-5)
- Skin rash—vesicular or vesiculobullous lesions/superficial desquamation
- Uveitis/chorioretinitis

Late findings include:

- Hutchinson triad
  - Hutchinson teeth
  - 8th nerve deafness
  - Interstitial keratitis
- Mulberry molars
- Saddle nose
- Clutton joints
- Bowing of the shins

Because of these complexities, the CDC has come up with different scenarios to help sort this out:

1) Infants with proven or highly likely disease have:
   A. An abnormal exam, or
   B. 4-fold or greater serum VDRL or RPR than the mother's, or
   C. Positive dark-field or fluorescent antibody test of body fluid.

For these infants, the following are recommended:

- CSF for VDRL, cell count, and protein
- CBC
- Other tests, if clinically indicated: long-bone x-rays, CXR, liver functions, cranial ultrasound, eye and hearing screens

Treat infants in this category of proven or highly likely congenital syphilis with:

Aqueous crystalline penicillin G 50,000 units/kg/dose IV q 12 hours during the first 7 days of life, then q 8 hours thereafter to complete 10 days, or procaine penicillin 50,000 units/kg/dose IM q day x 10 days.

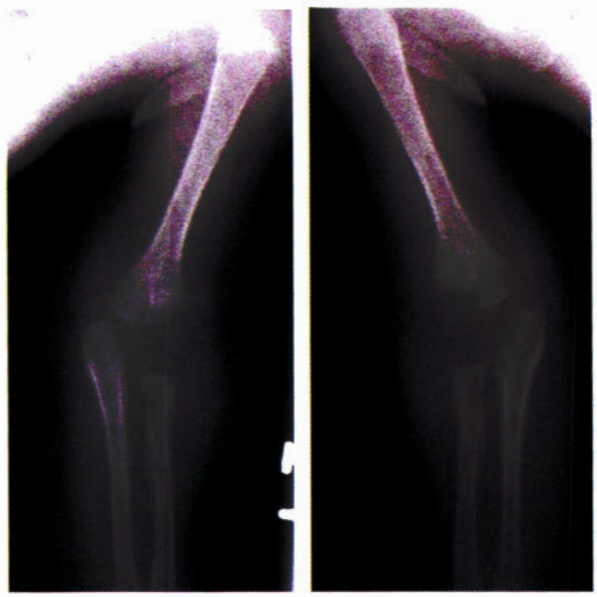
If you miss > 1 day of therapy, you have to restart at day 1.

2) Infants with normal physical examination and a serum-quantitative, nontreponemal-serologic titer the same or < 4x the mother's titer, and one of the following findings:
   A. Mother was not treated, or treatment status is unknown, or
   B. Mother was not treated with penicillin, or
   C. Mother was treated < 4 weeks before delivery.

For these infants the following are recommended:

- CSF for VDRL, cell count, and protein
- CBC
- Long bone x-rays

Treatment of this set of patients is varied and slightly controversial: Most recommend treating with the same as "proven" above, although some say a single



(Courtesy of The Journal of Bone and Joint Surgery)

*Image 4-5: Pseudoparalysis — Radiographs of the left and right upper extremities demonstrating methaphyseal erosions of the distal part of the humerus and the proximal part of the ulna.*

dose of benzathine penicillin G 50,000 units/kg/dose IM is acceptable—if the evaluation (CSF, CBC, long-bone films) is normal.

3) Infants who have a normal physical examination and a serum-quantitative, nontreponemal-serologic titer the same or < 4x the maternal titer and all of the following are true:
   A. Mother was treated during pregnancy, and treatment was appropriate > 4 weeks before delivery; and
   B. Mother has no evidence of relapse or reinfection.

   For these infants, no further evaluation is necessary.

   Give these infants benzathine penicillin G 50,000 units/kg/dose IM x 1.

4) Infants with a normal physical examination and a serum-quantitative, nontreponemal-serologic titer the same or < 4x the maternal titer and:
   A. Mother's treatment was adequate before pregnancy, and
   B. Mother's nontreponemal serologic titer remained low and stable before and during pregnancy and at delivery (VDRL ≤ 1:2; RPR ≤ 1:4).

   These infants require no further evaluation.

   Treatment: No treatment is recommended; however, some specialists would treat with a single IM injection of penicillin.

Here are some congenital syphilis pictures that could show up on the Board exam:

- Hutchinson teeth (Image 4-6); note the notching
- Mulberry molar: The first lower molar is now "dome-shaped" due to congenital syphilis (Image 4-7).
- Syphilitic rhinitis ("snuffles"), highly infectious (Image 4-8)

Conclusion for neonatal syphilis: On the Board exam, if you are ever in doubt about whether to treat, just do it (treat)! It is likely the right answer 95% of the time ☺.

## Chancroid

Chancroid is much rarer than either herpes or syphilis but can occur in the U.S., especially in discrete outbreaks associated with poverty, urban prostitution, and illicit drug use. The infection is due to *Haemophilus ducreyi*, which must be cultured on special culture media (Image 4-9). There is no FDA-approved PCR test available for this organism. Patients present with painful ulcers (unlike syphilis) and inguinal lymphadenopathy (Image 4-10).

Diagnosis of chancroid can be made on a clinical basis:

- One or more painful genital ulcers surrounded by an erythematous halo are present. The ulcer is soft, friable, and often eroded and associated with a foul-smelling, yellowish-gray exudate.
- No evidence of syphilis by dark-field examination of ulcer exudate, or a serologic test for syphilis done at least 7 days after the onset of the ulcers.
- The clinical presentation is appearance of genital ulcers and regional lymphadenopathy, if present, is consistent.
- HSV testing was performed on the ulcer and is negative.

Painful genital ulcers and tender, suppurative, usually unilateral inguinal lymphadenopathy (known as a bubo) are suggestive for chancroid.

Treatment of chancroid:

- Azithromycin 1 gram x 1, or
- Ceftriaxone 250 mg IM x 1, or
- Ciprofloxacin 500 mg bid x 3 days, or
- Erythromycin 500 mg tid x 7 days

## Granuloma Inguinale (Donovanosis)

Granuloma inguinale is caused by *Klebsiella granulomatis* (formerly known as *Calymmatobacterium granulomatis*). It is rare in the U.S. It presents as a painless, friable, progressive ulcerative lesion without regional lymphadenopathy (Image 4-11). These lesions are extremely vascular and can easily bleed on contact. Diagnose by visualizing dark-staining Donovan bodies

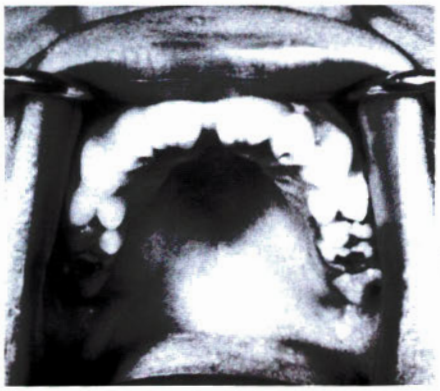

Courtesy of CDC Susan I Indsley

*Image 4-6: Hutchinson Teeth*

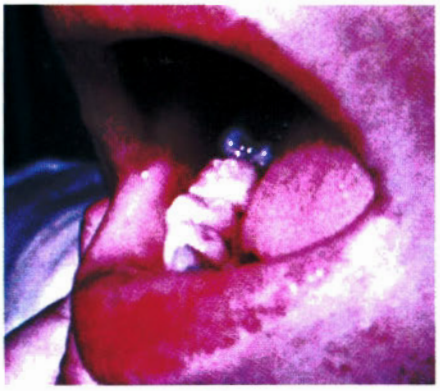

Courtesy of CDC Robert Kumpter

*Image 4-7: Mulberry Molar*

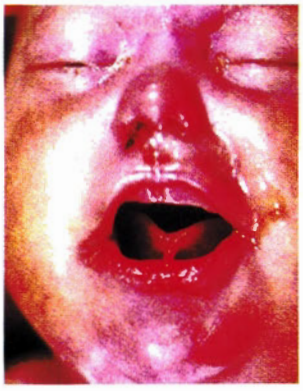

Courtesy of CDC Norman Cole, MD

*Image 4-8: "Snuffles"*

**QuickQuiz**

- What organism is responsible for chancroid?
- How does chancroid present, compared to syphilis?
- What is the organism responsible for granuloma inguinale?
- How does granuloma inguinale present?
- What organism is responsible for lymphogranuloma venereum?

(intracytoplasmic inclusion bodies) on tissue-crush preparation or biopsy.

Treatment: doxycycline 100 mg bid for a minimum of 3 weeks and until all lesions have healed.

### Lymphogranuloma Venereum

Lymphogranuloma venereum (LGV) is due to *Chlamydia trachomatis serovars* L1, L2, or L3 (the L's go with Lymphogranuloma). Patients present with a painless genital ulcer at the site of inoculation, followed by unilateral tender inguinal and/or femoral lymphadenopathy. The ulcer has usually disappeared before the patient seeks treatment. Use serology for diagnosis; a complement fixation titer ≥ 1:64 is consistent with LGV.

Treatment: doxycycline 100 mg bid x 21 days.

Alternative: erythromycin 500 mg qid x 21 days.

## STDs WITH URETHRITIS AND CERVICITIS

### Chlamydial Infections

#### Men

Urethritis in men is due to an infection that classically presents as a urethral discharge or mucopurulent/purulent material with occasional dysuria. Asymptomatic infections are common. The main 2 organisms to worry about in men are *Neisseria gonorrhoeae* and *Chlamydia trachomatis*. It is called "nongonococcal" urethritis (NGU) if gram-negative intracellular diplococci are not seen on urethral smears. *C. trachomatis* is the most common cause of NGU, but other organisms, such as *Ureaplasma urealyticum*, *Mycoplasma genitalium*, *Trichomonas vaginalis*, and herpes simplex, have been described. No symptoms or signs reliably distinguish gonococcal from NGU or among various etiologies of NGU. Make the diagnosis by Gram stain. If Gram stain shows ≥ 5 WBCs/oil field, this is highly sensitive and specific for urethritis. Also, you can do a leukocyte esterase test on first-void urine or microscopic examination of first-void urine for ≥ 10 WBCs/high power field. If these are not found, do specific tests for *N. gonorrhoeae* and *C. trachomatis*.

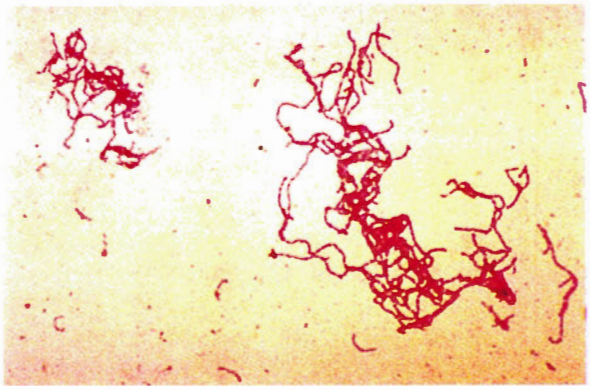
The CDC guidelines base diagnosis on finding at least 1 of the following:

1) Mucopurulent or purulent discharge
2) Gram stain of urethral secretions demonstrating > 5 WBC/hpf
3) Positive leukocyte esterase test on first-void urine or microscopic examination of first-void urine sediment demonstrating >10 WBC/hpf

Other available diagnostic tests include culture, direct immunofluorescence, ELISA, and nucleic acid amplification techniques (NAATs). The most sensitive and specific of these tests is NAATS, which can be performed on urethral specimens and on urine.

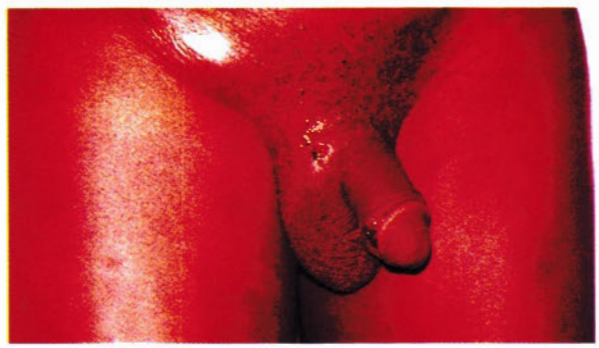
Treatment of *chlamydial* infections in men:

- Azithromycin 1 g x 1 dose, or
- Doxycycline 100 mg bid x 7 days



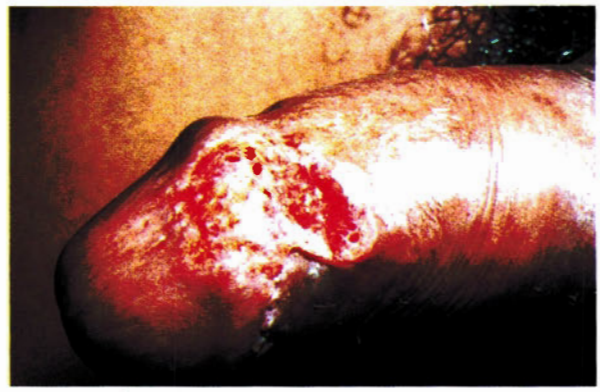
Courtesy of CDC: Greg Hammond, MD

*Image 4-9: Chancroid*



Courtesy of CDC: J. Pledger

*Image 4-10: Chancroid with Inguinal Lymphadenopathy*



Courtesy of CDC: Joe Miller, Cornelio Arevalo, MD

*Image 4-11: Granuloma Inguinale*

Alternative:

- Erythromycin 500 mg qid x 7 days, or
- Erythromycin ethylsuccinate 800 mg qid x 7 days, or
- Ofloxacin 300 mg bid x 7 days, or
- Levofloxacin 500 mg q day x 7 days
- (Remember: The quinolones are no longer effective for concomitant gonorrheal infections.) Don't forget to treat partners!

Some patients will have recurrent or persistent urethritis. Evaluate for *T. vaginalis*, and also ensure that any partners are treated. If you are assured of compliance and the partners have been treated, use the following for treatment:

- Metronidazole 2 g x 1 dose or tinidazole 2 g x 1 dose

plus

- Azithromycin 1 g x 1 dose

Approximately 1% of men with urethritis develop reactive arthritis, and approximately 1/3 of these patients have the complete manifestations formerly referred to as Reiter syndrome (arthritis, uveitis, and urethritis).

### Women

Mucopurulent cervicitis (MPC) is often asymptomatic, but some women have discharge or vaginal bleeding, especially after sexual intercourse. MPC can be due to *C. trachomatis*, *N. gonorrhoeae*, or neither. Test with culture or nucleic acid amplification procedures to identify the organism responsible. Also consider empiric therapy for a woman who has suspected gonorrhea or *Chlamydia* if the patient's risk factors suggest a high prevalence and there is a good likelihood that she will not return for therapy:

- Adolescents and young adults (age = biggest risk factor!)
- Multiple sex partners, a partner with other partners during the last three months, or a recent new sex partner
- Inconsistent use of barrier contraceptives
- Clinical evidence of mucopurulent cervicitis
- Cervical ectopy
- Unmarried status

Chlamydial infections in sexually active adolescents are quite common, and most are asymptomatic. Annually screen sexually active adolescent females, even those without symptoms. The concern is pelvic inflammatory disease (PID), increased risk of ectopic pregnancy, and infertility. Treat both *Chlamydia* and gonococcal infections. The CDC 2010 STD guidelines note that because cervicitis might be a sign of upper-genital tract infection (endometritis), women who seek medical treatment for a new episode of cervicitis should be assessed for signs of PID and should be tested for *C. trachomatis* and for *N. gonorrhoeae* with the most sensitive and specific test available. Women with cervicitis also should be evaluated for the presence of bacterial vaginosis (BV) and trichomoniasis, and if these organisms are detected, they should be treated.

Treatment for MPC:

- Azithromycin 1 g x 1 dose, or
- Doxycycline 100 mg bid x 7 days

Alternative treatment for MPC:

- Erythromycin 500 mg qid x 7 days, or
- Erythromycin ethylsuccinate 800 mg qid x 7 days, or
- *Chlamydia* only!: Ofloxacin 300 mg bid x 7 days, or
- *Chlamydia* only!: Levofloxacin 500 mg q day x 7 days
- (Remember: The quinolones are no longer effective for concomitant gonorrheal infections.)

Many believe that adolescents should be retested in 3–4 months to look for recurrence.

Treatment of MPC in pregnant women:

- Azithromycin 1 g x 1, or
- Amoxicillin 500 mg tid x 7 days

Alternative:

- Erythromycin 500 mg qid x 7 days, or
- Erythromycin ethylsuccinate 800 mg qid x 7 days, or
- Erythromycin ethylsuccinate 400 mg qid x 14 days

Note: Erythromycin estolate is contraindicated in pregnancy due to increased risk of hepatotoxicity. Also, always have the pregnant women return for follow-up testing to assess for cure.

## Gonorrheal Infections

Most men are symptomatic, while most women are not. Gonorrheal infections in women can cause the same complications as *Chlamydia* infections, including PID, increased risk of ectopic pregnancy, and infertility. Usually, treatment for gonorrhea also includes therapy for *Chlamydia*. Gonorrhea treatment is complicated by the ability of *N. gonorrhoeae* to develop resistance to antimicrobial therapies. Quinolone-resistant *N. gonorrhoeae* strains are now widely disseminated throughout the United States. This emergence of resistance led the CDC to discontinue recommending any fluoroquinolone regimens for the treatment of gonorrhea in 2007. In July 2011, the CDC released data from the Gonococcal Isolate Surveillance Project (GISP) showing that the percentage of isolates with elevated minimum inhibitory concentrations (MICs) to cephalosporins increased from 0.2% in 2000 to 1.4% in 2010 for cefixime and from 0.1% in 2000 to 0.3% in 2010 for ceftriaxone. The CDC now recommends dual therapy for gonorrhea infections of the cervix, urethra, and rectum with a cephalosporin plus azithromycin in hopes that routine co-treatment

**QuickQuiz**

- Know the main treatments for all of the STDs.
- How often should you screen an asymptomatic adolescent who is sexually active for STDs?
- For every STD, know the treatment regimen for pregnant women!
- What is Fitz-Hugh-Curtis syndrome?

might hinder the development of further antimicrobial-resistant *N. gonorrhoeae*.

From the 2010 CDC guidelines: Specific diagnosis of infection with *N. gonorrhoeae* can be performed by testing endocervical, vaginal, urethral (men only), or urine specimens. Culture, nucleic acid hybridization tests, and NAATs are available for the detection of genitourinary infection with *N. gonorrhoeae*.

The following is recommended for uncomplicated gonococcal infections of the cervix, urethra, and rectum: ceftriaxone 250 mg IM x 1 + azithromycin 1 g PO x 1

For treatment of uncomplicated gonococcal infections of the pharynx:

- Ceftriaxone 250 mg IM x 1
  plus, if *Chlamydia* has not been ruled out:
- Azithromycin 1 g PO x 1, or
- Doxycycline 100 mg PO bid x 7 days

Always think of gonococcal disease in acute pharyngitis in a sexually active adolescent!

Pregnant women: Do not use a quinolone or tetracycline. Use a cephalosporin for gonorrhea and either erythromycin or amoxicillin for *C. trachomatis*. If she is allergic to cephalosporins, the 2010 CDC guideline recommends azithromycin 2 g PO x 1.

## DISSEMINATED GONOCOCCAL INFECTION

This is a classic Board topic. [Know!] Disseminated gonococcal infection (DGI) accompanies bacteremia. It occurs in 1–3% of individuals with gonorrhea and is most common in females within a week of their last menses. Other risk factors include the peripartum period, SLE, and complement deficiencies. It can present as petechial or pustular skin lesions, asymmetrical arthralgia, tenosynovitis, or frank septic arthritis. See Image 4-12, with left knee showing arthritis with a skin lesion over the knee.

DGI may present in 1 of 2 ways, with very different means of diagnosing:

1) Symptoms of tenosynovitis and dermatitis associated with fever, chills, skin lesions, polyarthralgia (hands, wrists, fingers), positive blood cultures (30–40%), and negative synovial cultures
2) Suppurative monoarticular arthritis (knee usually) associated with positive synovial culture (45–55%) and negative blood culture

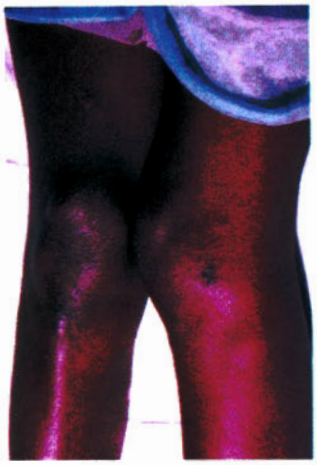


*Image 4-12: GC Septic Arthritis*

So, for the DGI with skin lesions and polyarthralgia, always check cervical and blood cultures (do not tap the joint); and for the DGI without skin lesions but with a monoarticular arthritis, always tap the joint (usually it's the knee)!

It can also present as perihepatitis (Fitz-Hugh-Curtis syndrome—look out for the adolescent female with upper-right quadrant abdominal pain!). More about this in the Infectious Disease section.

Treatment of disseminated gonococcal infection: ceftriaxone 1 gm IM/IV q 24 hours.

Alternatives:

- Cefotaxime 1 g IV q 8 hours, or
- Ceftizoxime 1 g IV q 8 hours, or
- Spectinomycin 2 g IM q 12 hours

Continue the above for 24–48 hours, until you see improvement; then you might change treatment to 1 of the following to complete at least 1 week of therapy:

- Cefixime 400 mg bid, or
- Cefpodoxime 400 mg PO bid

Usually we treat for concomitant *Chlamydia* as well.

If meningitis occurs, use ceftriaxone 1–2 g IV q 12 x 10–14 days. For endocarditis, prescribe 4 weeks of therapy at the same dosages.

## INFECTIONS WITH VAGINAL DISCHARGE

### Overview

3 diseases most commonly cause vaginal discharge:

1) Bacterial vaginosis (due to replacement of normal flora)
2) Trichomoniasis (due to *Trichomonas vaginalis*)
3) Candidiasis (usually due to *Candida albicans*)

Do pH testing to determine the etiology of the infection. Bacterial vaginosis and trichomoniasis usually elevate the pH to > 4.5. Presence of an amine odor after adding KOH to the discharged material indicates bacterial

vaginosis. You will see *T. vaginalis* as motile organisms on a wet mount and clue cells for bacterial vaginosis. Yeasts are visible on KOH for candidal infection.

## Bacterial Vaginosis

Bacterial vaginosis (BV) is a clinical syndrome due to the replacement of the normal flora, particularly *Lactobacillus*, in the vagina. The *Lactobacillus* is frequently replaced by anaerobes (*Prevotella* and *Mobiluncus*), *Gardnerella vaginalis*, and *Mycoplasma hominis*. **BV is the most common cause of vaginal discharge.** Treatment of male sexual partners is **not** beneficial. Although 50–75% of cases may be asymptomatic, a thin, homogenous, grayish-white discharge, often more noticeable after intercourse and associated with a pungent "fishy" odor, may be noted.

Clinical diagnosis requires 3 of the 4 following findings:

1) Homogenous, white, noninflammatory discharge that smoothly coats the vaginal walls
2) Clue cells (epithelial cells that appear granular and stippled with ragged "moth-eaten" borders) on microscopic examination (the single most reliable predictor of bacterial vaginosis)
3) pH of vaginal fluid is > 4.5
4) Fishy odor of vaginal discharge before or after the addition of 10% KOH

Gram stain to determine the relative concentration of lactobacilli, gram-negative and gram-variable rods and cocci (*G. vaginalis*, *Prevotella*, etc.), and curved gram-negative rods (*Mobiluncus*) is the "gold standard" laboratory method of diagnosing BV. Culture of *G. vaginalis* is not recommended because it is not specific. Newer DNA probes are available but are not recommended by the CDC at this time.

Treatment of bacterial vaginosis:

- **Metronidazole** 500 mg bid x 7 days, **or**
- Metronidazole gel 0.75%, one full applicator intravaginally, q day x 5, **or**
- Clindamycin cream 2%, one full applicator intra-vaginally at bedtime x 7 days

Alternatives:

- Clindamycin 300 mg bid x 7 days, **or**
- Clindamycin ovules 100 g intravaginally at bedtime x 3 days, **or**
- Tinidazole 2 g PO daily x 2 days or 1 g PO daily x 5 days

Pregnant women:

- Metronidazole 500 mg bid x 7 days, **or**
- Metronidazole 250 mg tid x 7 days, **or**
- Clindamycin 300 mg bid x 7 days

We know that BV in asymptomatic pregnant women at high risk for preterm delivery (those with a previous preterm infant) is associated with abnormal pregnancy outcomes (premature rupture of membranes, chorioamnionitis, preterm labor, preterm birth, post-cesarean wound infection); so far, topical therapy is not effective in pregnancy. Risk of teratogenicity with the use of metronidazole in pregnancy has recently been disputed.

Treatment of **asymptomatic** pregnant women at **low risk** for preterm delivery is more controversial—data are unclear if it makes any difference in incidence of preterm delivery, and a few studies suggest that intravaginal clindamycin may actually increase risk of adverse events.

## Trichomoniasis

Trichomoniasis is due to the protozoa *Trichomonas vaginalis*. Most men are asymptomatic, while most women have symptoms; e.g., diffuse, bubbly, or frothy malodorous yellow-green discharge with dysuria, pruritus, and vulvar irritation. Cervicitis ("strawberry cervix") is common. Asymptomatic infection also can occur in women. You usually can see the organisms on wet mounts, or they can be cultured (Image 4-13). Treatment of sexual partners is recommended.

Treatment of trichomoniasis:

- **Metronidazole** 2 g x 1 dose, **or**
- Tinidazole 2 g x 1 dose

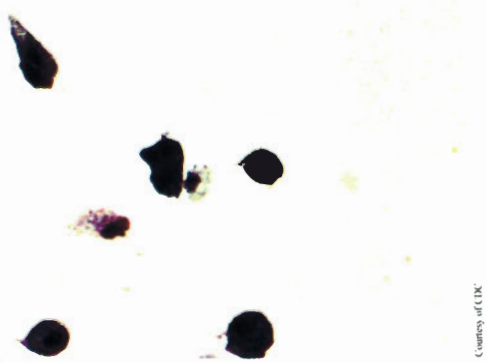
Alternative: metronidazole 500 mg bid x 7 days

Recently, treatment failure has become more common, and most recommend retreating with metronidazole 500 mg bid x 7 days or tinidazole 2 g x 1 dose. If retreatment fails, use 2 g of either agent q day x 5 days.

Give pregnant women metronidazole 2 g x 1 dose.

## Vulvovaginal Candidiasis

Vulvovaginal candidiasis is usually due to *Candida albicans*, but other candidal species can be involved. Typical symptoms include itching, intense burning, and whitish curd-like vaginal discharge. Most women



Courtesy of CDC

*Image 4-13: Trichomonas vaginalis*

**Quick Quiz**

- What are the characteristics of bacterial vaginosis infection?
- Should you treat male partners of a female with bacterial vaginosis?
- How is trichomoniasis treated? How do you treat it in a pregnant woman?
- Name all the treatments for vulvovaginal candidiasis. (Just kidding.) Name the only approved oral therapy.

will have at least 1 episode of vulvovaginal candidiasis during their lifetime, and most of these will be uncomplicated. Complicated vulvovaginal candidiasis is defined as recurrent, severe non-albicans infection; or in women with uncontrolled diabetes, debilitation, immunosuppression (including HIV), or pregnancy. Another common risk factor is recent antibiotic or oral contraceptive use.

Diagnose uncomplicated vulvovaginal candidiasis by doing a wet prep or Gram stain or culture for the organism. *Candida* vaginitis is usually associated with a pH < 4.5. KOH is very helpful. Look for budding yeast and pseudohyphae.

Treatment of uncomplicated vulvovaginal candidiasis: (Okay … Please do not memorize all of these for the Board exam!)

Intravaginal agents (use any of the following):

- Butoconazole 2% cream 5 g intravaginally x 3 days
- Butoconazole 2% cream 5 g sustained release x 1 dose
- Miconazole 2% cream 5 g intravaginally x 7 days
- Miconazole 100 mg vaginal suppository x 7 days
- Miconazole 200 mg vaginal suppository x 3 days
- Miconazole 1,200 mg vaginal suppository x 1
- Nystatin 100,000 unit vaginal tablet x 14 days
- Tioconazole 6.5% ointment 5 g intravaginally x 1
- Terconazole 0.4% cream 5 g intravaginally x 7 days
- Terconazole 0.8% cream 5 g intravaginally x 3 days
- Terconazole 80 mg vaginal suppository, x 3 days

or

Oral agent: fluconazole 150 mg oral tablet x 1 dose

They won't ask you to differentiate between these, or to know that if you use the terconazole 0.8% cream, you need to do it for only 3 days. Just know that the topicals work, and 1 oral agent works. Male partners may need to be treated topically if there is recurrence in the female partner (controversial), or if the male has evidence of balanitis.

### Complicated Vulvovaginal Candidiasis

Recurrent vulvovaginal candidiasis is usually defined as having 4 or more recurrences/year. Be sure to consider HIV in these patients on the Board examination, especially if you can't seem to get rid of the candidal infection. Do vaginal cultures to try to identify an unusual species, such as *C. glabrata*. Usually, these patients require longer initial therapy of 7–14 days for topicals or a repeat oral dose of fluconazole 3 days later. Some of these women may require maintenance antifungals, which they can take once weekly as vaginal suppositories or oral agents (e.g., fluconazole, itraconazole). Generally, continue these for 6 months.

Severe vulvovaginitis is characterized by extensive vulvar erythema, edema, excoriation, and fissure formation. Here again, longer treatment duration is usually necessary and is the same as outlined above for recurrent vulvovaginal candidiasis.

Non-albicans vulvovaginal candidiasis can be difficult to treat. Longer duration of therapy with a non-fluconazole azole drug is recommended. If there is recurrence, use 600 mg boric acid in a gelatin capsule vaginally daily x 2 weeks.

Compromised hosts do not respond well to therapy. In these situations, encourage the patient to modify conditions as much as possible.

Pregnant patients frequently get vulvovaginal candidiasis, and only topical azole therapy x 7 days is recommended.

## PELVIC INFLAMMATORY DISEASE

Pelvic inflammatory disease (PID) is a catchall phrase for inflammatory disorders of the upper female tract, including any combination of endometritis, salpingitis, tubo-ovarian abscess, and pelvic peritonitis. Most of the time, organisms obtained through sexual intercourse are the culprits, including *N. gonorrhoeae* and *C. trachomatis*. But other normal vaginal flora have also been found to be responsible, including anaerobes, *Gardnerella vaginalis*, *Haemophilus influenzae*, enteric gram-negative rods, and group B streptococcus (*Streptococcus agalactiae*). CMV, *Mycoplasma hominis*, and *Ureaplasma urealyticum* also have been implicated.

Symptoms for PID vary, and it is sometimes a difficult diagnosis to make. Many women with PID have subtle or mild symptoms. Typically, patients complain of dull, steady, unilateral or bilateral lower abdominal and/or pelvic pain which varies in severity from indolent to excruciating. Additional symptoms may include fever, right-upper quadrant pain, vomiting, vaginal discharge, and irregular vaginal bleeding. Laparoscopy can be very helpful if you suspect salpingitis, but this diagnostic tool is usually not immediately accessible. Unfortunately, no single item in the history, physical, or laboratory is very sensitive or specific in making the diagnosis, and using

a combination of tests reduces either the sensitivity or specificity, making it less likely to be helpful. Many cases of PID are undiagnosed because of failure to recognize the mild/subtle symptoms of the disease in many women. Undiagnosed PID can cause sterility.

Because it is such a difficult diagnosis to make, the CDC has defined minimum criteria for making the diagnosis. These criteria are very sensitive but, unfortunately, not very specific. The important thing is to **not** miss a diagnosis of PID because of the potentially adverse, long-term outcomes (e.g., sterility, abscess). **Generally, you start empiric therapy for PID if a sexually active woman presents with 1 of these: uterine/adnexal tenderness or cervical motion tenderness—and you can't find another explanation.**

Other things can be helpful in making, or supporting, the diagnosis:

- Temperature > 101° F
- Tenderness to palpation of the lower abdomen with or without rebound/guarding
- Friable, inflamed cervix
- Abnormal cervical or vaginal mucopurulent discharge
- Presence of WBCs on saline microscopy of vaginal secretions
- Elevated ESR
- Elevated C-reactive protein (CRP)
- Documentation of cervical infection with *N. gonorrhoeae* or *C. trachomatis*

(Laboratory tests, such as ESR and CRP, may be helpful, but are not commonly done because they lack specificity.)

**Note**: If the cervical discharge is normal, and you find **no** WBCs on wet prep, the diagnosis of PID is unlikely.

The most **specific criteria** for diagnosing PID (but these are rarely done!) include:

- Endometrial biopsy for histopathologic evidence of endometritis
- Transvaginal sonogram or MRI to show thickened, fluid-filled tubes, with or without free pelvic fluid or tubo-ovarian complex
- Laparoscopic evidence of PID

Sometimes, some of the above are done if the diagnosis is in doubt or if certain aspects of the case warrant further evaluation. Sorry that this is vague, but, at least for the Pediatric Boards, you won't need to know the indications for these procedures. For the Boards, if there is any question that this might be PID, **treat**!

When do you hospitalize women with PID? This is also difficult, but know these general guidelines:

- **Surgical emergencies** cannot be excluded (appendicitis, ectopic pregnancy).
- Pregnancy.
- Clinical response to oral antimicrobial therapy is **inadequate**.
- The patient is unable to follow or tolerate an outpatient oral regimen. (Some would say most adolescents fall into this category, but actually no data are available that suggest that adolescents benefit from hospitalization for treatment of PID.)
- **Severe illness**, nausea, and vomiting, or high fever are present.
- You suspect or find a **tubo-ovarian abscess**.

Treatment of PID: Okay, here is the nightmare: There are **many** acceptable therapies for PID. Note that as of the publication of this text, no further changes were recommended to the 2010 CDC guidelines in particular for treating inpatient or outpatient PID with the combo therapy used above (ceftriaxone + azithromycin). Realize this may be in flux; however, commonly the ABP is very slow in updating its questions with new information. For the Boards, I would answer with the best answer that is valid at the time I take the test; if that response is not there, then I would go with the previous best answer—I would not spend time trying to second-guess the Boards on whether they are current or not. For example, if they asked me about treating uncomplicated cervical gonorrhea and they had an answer choice of ceftriaxone 250 mg IM x 1 + azithromycin 1 g PO x 1, I would pick that (because that is the current recommended therapy as of July 2011); alternatively, if the only choice they had was ceftriaxone 250 mg IM x 1 without the azithromycin (the current recommended therapy in the 2010 CDC guidelines), then I would pick that choice. They are not trying to trick you—but updates happen all the time, so go with the answer choice that is most current, if that choice is listed. If it is controversial, the Boards will throw out the question.

Parenteral treatments for PID (first-line):

- Cefotetan 2 g IV q 12 hours, **or**
- Cefoxitin 2 g IV q 6 hours

**plus**

- Doxycycline 100 mg orally or IV q 12 hours

You can stop parenteral therapy after the patient has shown improvement for 24 hours; then complete 14 days of doxycycline.

Another parenteral protocol for PID:

- Clindamycin 900 mg IV q 8 hours

**plus**

- Gentamicin-loading dose of 2 mg/kg IV/IM, followed by maintenance dose of 1.5 mg/kg q 8 hours. (You can also use single, daily dosing.) For Boards, use this in someone who is cephalosporin-allergic!

**QuickQuiz**

- Describe when to hospitalize a patient with PID.
- Name several PID treatments.
- What causes epididymitis in adolescent males?
- Which HPV types can predispose to cancer?

Still more alternative therapies for PID (least used):

- Ampicillin/sulbactam 3 g IV q 6 hours

plus

- Doxycycline 100 mg PO or IV q 12 hours

After improvement has occurred, stop the parenteral therapy and use doxycycline 100 mg bid, or clindamycin 450 mg qid to complete 14 days of therapy.

What about outpatient therapy?

Outpatient treatments for PID:

- Ceftriaxone 250 mg IM x 1, or
- Cefoxitin 2 g IM x1, and probenecid 1 g PO x 1, or
- Other 3rd generation cephalosporins

plus

- Doxycycline 100 mg bid x 14 days

with or without

- Metronidazole 500 mg bid x 14 days

For outpatient therapy, if no clinical improvement occurs within 72 hours, hospitalize the patient and place on a parenteral regimen.

Don't forget to treat PID patients' partners!

## EPIDIDYMITIS

For adolescent males, the most common cause of epididymitis is an STD due to *C. trachomatis* or *N. gonorrhoeae*. You occasionally see gram-negatives, such as *Escherichia coli* in males who engage in anal intercourse. Treat most as outpatients unless there is severe pain or complication, or you are uncertain of the diagnosis (testicular torsion, infarction). Most adolescents present with unilateral testicular pain and tenderness, with findings of hydrocele and palpable swelling. Onset is most commonly gradual but can be sudden. Adolescents in particular are prone to testicular torsion, so you should always consider it in the differential diagnosis. If you suspect testicular torsion, immediately refer the patient to an emergency department where a urologist can treat the patient.

For diagnostic purposes, do one of the following:

- Gram-stained smear of urethral exudate, or intraurethral swab to look for ≥ 5 PMNs/oil immersion field and *N. gonorrhoeae*; i.e. gram-negative intracellular diplococci!
- Intraurethral exudate culture, or a nucleic acid amplification test (either swab or first-void urine) for *N. gonorrhoeae* and *C. trachomatis*.
- Leukocyte esterase test on first-void urine or microscopic examination of first-void urine sediment showing ≥ 10 WBC/hpf.

Syphilis and HIV testing should be done on all suspected/confirmed cases.

Treatment for epididymitis: Always start empiric antibiotics pending laboratory results. Also recommend bed rest and scrotal elevation, and treat for pain relief.

If the epididymitis is most likely due to gonococcal or chlamydial infection (as it will be in a majority of adolescents):

- Ceftriaxone 250 mg IM x 1

plus

- Doxycycline 100 mg bid x 10 days

If, for some reason, you suspect an enteric gram-negative organism is responsible:

- Ofloxacin 300 mg bid x 10 days, or
- Levofloxacin 500 mg q day x 10 days

## HUMAN PAPILLOMAVIRUS INFECTION (HPV)

There are > 40 different types of human papillomavirus (HPV) that can infect the genital tract. Most infections are asymptomatic and subclinical. HPV types 6 and 11 usually cause visible warts. Besides the genital area, HPV types 6 and 11 can produce warts in the conjunctival, nasal, oral, and laryngeal areas. In affected infants, laryngeal papillomatosis presents with hoarseness, altered cry and, at times, stridor. The lesions typically recur soon after removal and many patients require repeated surgeries to control symptoms and prevent respiratory compromise.

The HPV types we're most concerned about are those that are strongly associated with cervical neoplasia, and those that also can be found with squamous intraepithelial neoplasia of the penis, anus, and vulva, as well as squamous cell carcinoma. These are HPV types 16, 18, 31, 33, and 35. You probably should memorize these. (One way I remember is to think—well, at 16 and 18, most teenagers are "bad" and 31, 33, and 35 are "bad" because they are odd years early in your 30s.)

The warts due to HPV can occur on the penis, vulva, scrotum, perineum, perianal skin, vagina, urethra, anus, and/or mouth. Intraanal warts occur in those who have had receptive anal intercourse. Perianal warts, on the other hand, can occur in both men and women without a history of anal sex.

Aim treatment at removing symptomatic warts. Unfortunately, treatment does not eliminate infectivity of the virus. No particular one of the available treatments is superior to the others. In general, warts on moist surfaces and/or in intertriginous areas respond better to topical treatment, as opposed to warts on drier surfaces, which don't usually respond very well to topicals. Change treatments if there is not great improvement after 3 provider-given treatments, or if the warts have not completely cleared by 6 treatments.

Recommended regimens for external genital warts:

- Patient-applied:
  - Podofilox 0.5% solution or gel—apply bid for 3 days then off for 4 days, or
  - Imiquimod 5% cream once daily at bedtime, 3 times a week for up to 16 weeks, or
  - Sinecatechins 15% ointment 3 times a day for up to 16 weeks
- Provider-applied:
  - Cryotherapy with liquid nitrogen or cryoprobe, or
  - Podophyllin resin 10–25%, or
  - Trichloroacetic acid (TCA) or bichloracetic acid (BCA) 80–90%, or
  - Surgical removal

Alternatives include: intralesional interferon, topical cidofovir, or laser surgery.

Recommended regimens for vaginal warts:

- Cryotherapy with liquid nitrogen, or
- TCA or BCA 80–90%

Recommended regimens for urethral meatus warts:

- Cryotherapy with liquid nitrogen, or
- Podophyllin 10–25%

Recommended regimens for anal warts:

- Cryotherapy with liquid nitrogen, or
- TCA or BCA 80–90%, or
- Surgical removal

Recommended regimens for oral warts:

- Cryotherapy with liquid nitrogen, or
- Surgical removal

Pregnancy Issues: Do not use imiquimod, sinecatechins, podophyllin, or podofilox during pregnancy. HPV can cause recurrent laryngeal papillomatosis in infants and children, and, therefore, most recommend removal of genital warts if they occur during pregnancy. Do not perform a cesarean delivery solely to prevent HPV infection in the newborn. However, cesarean delivery may be indicated if genital warts are obstructing the pelvic outlet, or if vaginal delivery would result in excessive bleeding.

Subclinical Genital HPV Infection: Note: DNA or RNA tests for screening subclinical genital HPV infection are not recommended for adolescents and women under the age of 30. This is because HPV infection is highly prevalent and usually transient in women at younger ages, while the prevalence of cervical cancer is relatively low compared to older women. In the absence of genital warts or cervical squamous intraepithelial lesions, treatment is not recommended for subclinical genital HPV infection, whether it is diagnosed by colposcopy, biopsy, acetic acid application, or through detection of HPV by laboratory tests. If coexistent squamous intraepithelial lesions are present, treatment management depends on histopathologic findings.

## CERVICAL CANCER SCREENING

Current guidelines from the USPSTF and ACOG recommend that cervical screening begin at age 21 years. This recommendation is based on the low incidence of cervical cancer and limited utility of screening in younger women. The American Cancer Society recommends that women start cervical screening with Pap tests after 3 years of initiating sexual activity but by no later than age 21 years. Most follow the former recommendations, so in general we won't be doing Pap smears in pediatrics.

However, if a high-risk adolescent has an abnormal Pap, then the 2010 CDC guidelines note that the prevalence of HPV is high among adolescents < 21 years of age. Infections in adolescent patients tend to clear rapidly, and lesions caused by these infections also have high rates of regression to normal. Therefore, adolescents with atypical squamous cells of undetermined significance (ASC-US) or low-grade squamous intraepithelial lesions (LSIL) can be managed with repeat cytologic testing at 12 months and 24 months. Only those with high-grade squamous intraepithelial lesions (HSIL) at either follow-up visit or persistence of ASC-US or LSIL at 24 months should be referred for colposcopic evaluation. (HSIL includes moderate dysplasia/cervical intraepithelial neoplasia [CIN] 2, severe dysplasia/CIN 3, and carcinoma *in situ*.)

HPV DNA testing is not recommended as an adjunctive to the Pap—and is definitely not recommended for primary screening in women < 30 years of age.

## VACCINE-PREVENTABLE STDs

### Overview

Vaccines are currently available to prevent HPV, hepatitis A, and hepatitis B, the latter two of which are both potential sexually transmitted diseases. Trials are underway to investigate vaccines for HIV and herpes. Know: When you see an adolescent (especially on the Board exam), offer her/him hepatitis B vaccination if she/he is unvaccinated. HPV vaccine is recommended for all 9–26 years of age. Give the hepatitis A vaccine to adolescent men who have sex with men, and adolescents who abuse IV drugs (but don't forget this vaccine is now universally recommended for all children).

**QuickQuiz**

- List therapy for HPV.
- List therapy for HPV in a pregnant adolescent.
- Which adolescents should get hepatitis A vaccine?
- How do you protect someone who is exposed to active hepatitis A? hepatitis B?
- True or false? There is effective prophylaxis for hepatitis C exposure.
- What is contraindicated as therapy for lice in a pregnant woman?
- Name an effective therapy for scabies.
- What are the organisms responsible for scabies and lice?

### Hepatitis A

If an unvaccinated child ≥ 1 year of age is exposed through household or sexual contact, or by sharing IV drugs with a person with hepatitis A, give hepatitis A vaccine as prophylaxis! This is a new recommendation and is preferred over IM immune globulin. Now a single IM dose of immune globulin (0.02 mL/kg) is only recommended for those < 12 months of age or > 41 years of age.

### Hepatitis B

If an unvaccinated person is exposed through sexual contact or by sharing IV drugs with a person with hepatitis B, give hepatitis B immune globulin (HBIG) and hepatitis B vaccine—at separate sites at the same time—optimally within 24 hours, 7 days max for percutaneous exposure, and within 14 days for sexual contact exposure. For children and adolescents with household exposure, vaccinate with hepatitis B vaccine. HBIG is not necessary. For victims of sexual abuse, where the offender is not known to have acute hepatitis B, HBIG is not required, but give the vaccination.

### Hepatitis C

No vaccine is available for hepatitis C, and prophylaxis with immune globulin is not effective in preventing HCV infection.

### HPV

Two HPV vaccines are licensed in the United States: a bivalent vaccine (HPV2, Cervarix®) containing HPV types 16 and 18 and a quadrivalent vaccine (HPV4, Gardasil®) vaccine containing HPV types 6, 11, 16, and 18. Both vaccines protect against HPV types responsible for 70% of cervical cancers (16, 18). HPV4 also protects against HPV types that cause 90% of genital warts (6, 11). HPV vaccination (HPV4 or HPV2) is recommended for all girls 9–26 years of age. HPV4 can be given to boys 9–26 years of age as well to prevent genital warts. The vaccines have no therapeutic impact on disease caused by infection prior to vaccination. However, remember that vaccination is still indicated with a prior history of HPV, because it is highly unlikely that an individual was previously infected by all 2 or 4 HPV types contained in the vaccines.

## PROCTITIS, PROCTOCOLITIS, AND ENTERITIS

These can be sexually transmitted entities, depending upon an individual's sexual practices.

Proctitis is inflammation limited to the rectum (the distal 10–12 cm) and is usually associated with anorectal pain, tenesmus, or rectal discharge. The most common pathogens are usually *N. gonorrhoeae*, *C. trachomatis*, *T. pallidum*, and HSV. It is mainly seen in those who participate in receptive anal intercourse. The recommended treatment regimen for these patients is ceftriaxone 125 mg IM plus doxycycline 100 mg bid x 7 days.

Proctocolitis has similar symptoms to proctitis, but patients also have diarrhea, abdominal cramps, and inflammation of the colonic mucosa. Organisms causing this are likely to be *Campylobacter*, *Shigella*, and *Entamoeba histolytica*. Proctocolitis can be acquired by the oral or fecal-oral route. Aim treatment at the offending agent.

Enteritis presents as diarrhea and abdominal cramping without signs of proctitis or proctocolitis. It can be seen in those with sexual practices that include oral-fecal contact. *Giardia lamblia* is the most common pathogen identified. Treat with metronidazole.

## ECTOPARASITIC INFECTIONS

### Note

Lice and scabies can be sexually transmitted—and must be included in the differential for an adolescent presenting with persistent pruritus or nits.

### Pediculosis

Patients will usually present with itching because of the presence of nits (eggs) or lice in their scalp, body, or pubic hair. Nits appear as yellowish-white, glistening, oval particles attached to the hair shafts, while lice are tan to grayish-white in appearance. They are most often identified behind the ears or along the nape of the neck. See Image 4-14 for a photo of *Phthirus pubis* (crab louse). Resistance to common therapeutic agents is emerging rapidly in some areas.

Treatment of pediculosis includes applying:

- **Permethrin—1% lotion** (Nix®) **or 5% cream** (Acticin®, Elimite®)—is the drug of choice. 1% lotion is applied to affected areas for 10 minutes; 5% cream is applied for several hours to overnight.

  **or**

- Pyrethrins with piperonyl butoxide (RID®, A-200®, Pronto®, Clear®) for 10 minutes.

A repeat application of either agent is recommended by the CDC because of emerging resistance.

In mid-2009, benzoyl alcohol 5% was approved for treatment of head lice.

Alternatives:

- Malathion lotion 0.5% (Ovide®) is available by prescription but requires leaving on for 8–12 hours before washing off. It has not been evaluated in children < 6 years of age and is contraindicated in children < 2 years.
- Spinosad (Natroba™ Topical Suspension 0.9%) was approved by the FDA in 1/2011 for head lice treatment in children > 4 years.

Lindane 1% shampoo is no longer recommended. You may still see it listed in a test question but likely it is a distractor and you should **not** pick it.

Do **not** apply these body treatments to the eyes.

Treat eyelash involvement with occlusive ophthalmic ointment to the eyelid margins bid x 10 days. All bedding and clothing must be decontaminated (machine washed and machine dried using heat; or dry cleaned). Retreatment may be necessary if you observe lice or eggs at the hair/skin junction. Sexual partners within the last month should be treated.

For cases resistant to all forms of topical therapy, ivermectin 250 µg/kg PO x 1 dose with a repeat dose 2 weeks later can be effective. (Do not use in pregnant or lactating women or children < 15 kg.)

The AAP head lice statement notes that a single oral dose of 200 µg/kg of ivermectin, repeated in 10 days, has been shown to be effective against head lice. Most recently, a single oral dose of 400 µg/kg repeated in 7 days has been shown to be more effective than 0.5% Malathion lotion. (This is an off-label use of ivermectin.)

Most common causes of treatment failure are lack of compliance and continued contact with others who are infested and untreated. Unfortunately, many school districts do not allow children to return to school with nits in place—commonly known as the "no-nits" policy. "Stand alone" nits are not indicative of active infection. "No-nit" policies have **not** been shown to stop the spread of head lice and the AAP and CDC recommend that these policies are not appropriate.

## Scabies

Scabies requires sensitization to the organism, *Sarcoptes scabiei* (Image 4-15). The first time a person is infested with *S. scabiei*, it may take weeks before pruritus develops; however, on the next exposure, itching will occur within 24 hours. Burrows in the webs of the fingers and toes are common; the pruritus is **intense** (Image 4-16).

Treatment for scabies: Apply permethrin 5% cream to all areas of the body from the neck down, and wash off after 8–14 hours.

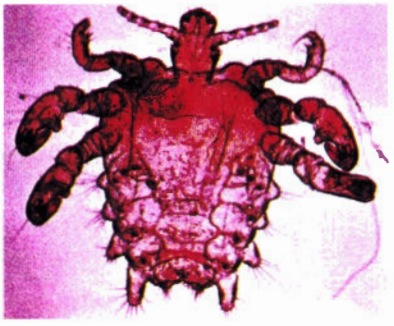
Alternative treatments are:

- Ivermectin 200 µg/kg PO, repeated in 2 weeks.
- "Last-resort alternative" is lindane 1% (but many avoid lindane because of neurotoxicity): Apply 1 oz of lotion or 30 grams of cream in a thin layer to all areas of the body from the neck down and thoroughly wash off after 8 hours. Do not apply lindane after a bath, or to someone with extensive atopic dermatitis, because **seizures** have been reported. Aplastic anemia has also been reported after use of lindane.

Sexual partners and close personal contacts within the last month should be examined and treated.

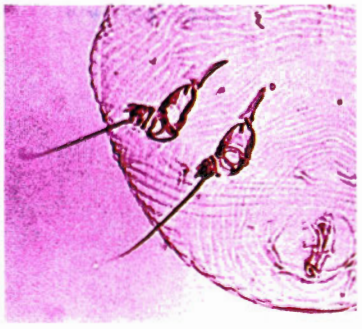
Decontaminate bedding and clothing, as with lice above.

The rash and itching of scabies may persist for up to 2 weeks after treatment.



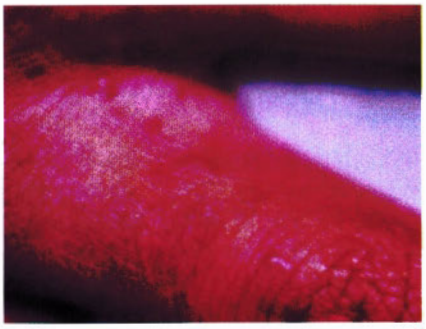
Courtesy of CDC

*Image 4-14: Phthirus pubis, "Crab Louse"*



Courtesy of CDC/ World Health Organization

*Image 4-15: Sarcoptes scabiei*



Courtesy of CDC

*Image 4-16: Scabies*

**Table 4-10:** Substance Abuse and Key Clinical Findings/Effects—1 of 2

| Drug | Symptoms/Signs | Lab/Other Effects |
|---|---|---|
| Marijuana | Impairment of short-term memory<br>Decreased performance in driving<br>Loss of critical judgment<br>Time perception distortion | Plasma testosterone levels decreased as well as spermatogenesis<br>Decreased glucose tolerance<br>Antiemetic effect<br>Significant drop in intraocular pressures (good for glaucoma)<br>Gynecomastia |
| Tobacco | Chronic cough, phlegm, and wheezing | Increased hemoglobin/hematocrit<br>Increased platelet aggregation |
| Toluene (airplane glue) | Relaxation and "pleasant" hallucinations for 2 hours | Excreted rapidly in the urine and may be detected by looking for hippuric acid |
| Gasoline | Euphoria followed by violent excitement and then coma | Pulmonary hypertension<br>Restrictive lung defects<br>Peripheral neuropathy<br>Acute rhabdomyolysis<br>Hematuria |
| Amyl nitrite ("poppers")<br>Butyl nitrite (room deodorizers) | Euphoria<br>Enhanced musical appreciation<br>Aphrodisiacs<br>Headaches<br>Syncope | Hypotension<br>Transiently inverted T waves and depressed ST segments<br>Methemoglobinemia<br>Increased intraocular pressure |
| LSD (lysergic acid diethylamide) | Very potent hallucinogen<br>3 stages:<br>1) Somatic (physical effects: dizziness, dilated pupils, flushing, increased temp, tachycardia)<br>2) Perceptual (changes in vision and hearing: "seeing smells" or "hearing" colors)<br>3) Psychic effects (changes in sensorium: delusional ideation, body distortion, psychosis) | Not associated with a withdrawal syndrome<br>"Flashbacks" common years after use |
| Alcohol | Euphoria<br>Grogginess<br>Talkativeness<br>Impaired short-term memory<br>High levels result in respiratory depression and death | Elevated GGT and AST |
| Inhalants | Euphoria<br>Slurred speech<br>Decreased coordination<br>Dizziness<br>Sexual enhancement (amyl nitrates)<br>Profound hypotension<br>Chronic use with neuromuscular disorders and restrictive lung disease and brain damage | Methemoglobinemia (amyl nitrate) with decreased oxygen saturation on pulse ox but normal $PaO_2$ |
| Dextromethorphan | "Out of body" dream-like state<br>Tachycardia, hypertension, lethargy, mydriasis, agitation, and vomiting | Symptoms last 3–6 hours |
| Mushrooms (psilocybin) | Similar to LSD | Also, prominent gastrointestinal symptoms; e.g., nausea, vomiting, and diarrhea |

**Table 4-11:** Substance Abuse and Key Clinical Findings/Effects—2 of 2

| Drug | Symptoms/Signs | Lab/Other Effects |
|---|---|---|
| MDMA (Ecstasy, methylenedioxymeth-amphetamine) | Euphoria, heightened sensual awareness<br>Increased "emotional" energy<br>Nausea, jaw clinching, teeth grinding, and blurred vision are common<br>Anxiety and psychotic episodes can occur | |
| PCP (phencyclidine, angel dust) | Cramps, diarrhea, and hematemesis<br>Euphoria, nystagmus, ataxia, and hallucination<br>Higher doses–psychosis with use of verbal abusive language<br>Even higher levels result in cardiac arrhythmias and seizures<br>Coma (unique for PCP) has absence of respiratory depression, presence of muscle rigidity, hyperreflexia, and nystagmus | Urine drug screen is helpful in diagnosis |
| Cocaine | Euphoria<br>Decreased fatigability<br>Paranoid ideation<br>Seizures can occur | Pupillary dilatation<br>Tachycardia<br>Hypertension<br>Consider in young person with MI: look for cocaine |
| Amphetamines | Hypertension<br>Hyperpyrexia<br>Seizures occur | Cardiac conduction abnormalities<br>Cerebrovascular damage<br>Withdrawal syndrome presents in stages:<br>Early: depression, agitation, anergia<br>Intermediate: loss of energy, limited interest in surroundings, anhedonia<br>Final: drug craving returns<br>Do not use phenothiazines for treatment of withdrawal! (hypotension and seizures) |
| Opiates | Euphoria<br>Decrease in pain<br>Pinpoint pupils | Severe vasodilation<br>Respiratory depression<br>Pulmonary edema<br>Loss of libido<br>Constipation<br>Withdrawal occurs 8 hours without heroin over a period of 24–36 hours: First finding seen is yawning; then lacrimation, mydriasis, insomnia, "goose flesh," diarrhea, and systolic hypertension. Diazepam helpful. |
| Anabolic steroids | Acne<br>Keloids<br>Stria<br>Hirsutism<br>Gynecomastia<br>Testicular atrophy<br>Azoospermia<br>Rage<br>Depression<br>Mania<br>Libido alterations | Hepatitis<br>Increased risk of hepatocellular carcinoma<br>Fluid retention is common<br>Growth retardation because of early epiphyseal closure |

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